

FILE 'HCAPLUS' ENTERED AT 13:30:24 ON 22 JUL 2008  
L1           3287 S URSOODEOXYCHOL?  
L2           139310 S ISCHEM? OR STROKE OR NEUROPROTECTIVE  
L3           28 S L1 AND L2  
L4           19 S L3 AND (PY<2005 OR AY<2005 OR PRY<2005)

FILE 'HCAPLUS' ENTERED AT 15:30:51 ON 22 JUL 2008  
L5           96359 S PREBIOTIC OR ENTERAL OR DIARRHEA OR NUTRITIONAL  
L6           493913 S ADHESIVE OR ADHESION

FILE 'HCAPLUS' ENTERED AT 15:31:22 ON 22 JUL 2008  
L7           95742 S ((ARABINO OR MANNO OR GALACTO OR ISOMALTO OR SIALYL) (W)OLIGOS  
L8           19 S L5 AND L6 AND L7

FILE 'HCAPLUS' ENTERED AT 15:32:11 ON 22 JUL 2008  
L9           8 S L8 AND (PY<2004 OR AY<2004 OR PRY<2004)

FILE 'HCAPLUS' ENTERED AT 15:51:07 ON 22 JUL 2008  
L10          2473 S HT29  
L11          1 S L5 AND L6 AND L10  
L12          1 S L5 AND L7 AND L10  
L13          0 S L11 AND (PY<2004 OR AY<2004 OR PRY<2004)  
L14          0 S L12 AND (PY<2004 OR AY<2004 OR PRY<2004)

FILE 'HCAPLUS' ENTERED AT 15:53:20 ON 22 JUL 2008  
L15          26 S L5 AND L10  
L16          188 S L6 AND L10  
L17          15 S L15 AND (PY<2004 OR AY<2004 OR PRY<2004)  
L18          115 S L16 AND (PY<2004 OR AY<2004 OR PRY<2004)

FILE 'REGISTRY' ENTERED AT 16:40:01 ON 22 JUL 2008  
EXP MANNOBIOSE/CN  
L19          1 S E4  
EXP ALPHA MANNOBIOSE/CN  
EXP ALPHA 1,3-MANNOBIOSE/CN  
EXP ALPHA 1,3 MANNOBIOSE/CN  
EXP 1,3 MANNOBIOSE/CN  
EXP ALPHA 1,2-MANNOBIOSE/CN  
EXP ALPHA 1,6-MANNOBIOSE/CN  
EXP MANNOOLIGOSACCH/CN

FILE 'CAPLUS' ENTERED AT 16:41:54 ON 22 JUL 2008  
L20          0 S L19/THU  
L21          1 S L19  
L22          380 S MANNOBIOSE

FILE 'REGISTRY' ENTERED AT 16:43:59 ON 22 JUL 2008  
L23          1 S MANNOBIOSE/CN

FILE 'CAPLUS' ENTERED AT 16:44:08 ON 22 JUL 2008  
L24          6 S L23/THU

FILE 'HCAPLUS' ENTERED AT 16:45:21 ON 22 JUL 2008  
L25          64 S METHYL ALPHA MANNO?  
L26          74681 S NUTRITIONAL OR ENTERAL OR PREBIOTIC  
L27          0 S L25 AND L26

FILE 'STNGUIDE' ENTERED AT 16:46:00 ON 22 JUL 2008

FILE 'HCAPLUS' ENTERED AT 16:46:41 ON 22 JUL 2008  
L28          714245 S GUT OR INTESTINE OR ORAL OR PHARMACEUTICAL

L29

4 S L25 AND L28

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FILE COVERS 1907 - 22 Jul 2008 VOL 149 ISS 4  
FILE LAST UPDATED: 20 Jul 2008 (20080720/ED)

HCAplus now includes complete International Patent Classification (IPC) reclassification data for the second quarter of 2008.

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s ((arabino or manno or galacto or isomalto or sialyl)(w)oligosaccharide)) or arabinooligosaccharide or mannooligosaccharide or galactooligosaccharide or isomaltooligosaccharide or sialyloligosaccharide or pectin or lactose or curdlan or lactulose or (beta(2a)glucan)

UNMATCHED RIGHT PARENTHESIS 'ACCHARIDE)) OR'  
The number of right parentheses in a query must be equal to the number of left parentheses.

=> s prebiotic or enteral or diarrhea or nutritional

4374 PREBIOTIC  
4337 ENTERAL  
22140 DIARRHEA  
66844 NUTRITIONAL

L5 96359 PREBIOTIC OR ENTERAL OR DIARRHEA OR NUTRITIONAL

=> s adhesive or adhesion

224861 ADHESIVE  
325612 ADHESION  
L6 493913 ADHESIVE OR ADHESION

=> file stnguide

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	2.69	64.26
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-15.20

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FILE CONTAINS CURRENT INFORMATION.  
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=> file hcaplus			
COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION	
FULL ESTIMATED COST	0.06	64.32	
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION	
CA SUBSCRIBER PRICE	0.00	-15.20	

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New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s ((arabino or manno or galacto or isomalto or sialyl)(w)oligosaccharide) or arabinoooligosaccharide or mannoooligosaccharide or galactooligosaccharide or isomaltoooligosaccharide or sialylooligosaccharide or pectin or lactose or curdlan or lactulose or (beta(2a)glucan)

4078 ARABINO  
2731 MANNO  
3051 GALACTO  
213 ISOMALTO  
4660 SIALYL  
32176 OLIGOSACCHARIDE  
289 (ARABINO OR MANNO OR GALACTO OR ISOMALTO OR SIALYL) (W)OLIGOSACCHARIDE  
10 ARABINOOLIGOSACCHARIDE  
213 MANNOOLIGOSACCHARIDE  
417 GALACTOOLIGOSACCHARIDE  
344 ISOMALTOOLIGOSACCHARIDE  
154 SIALYLOOLIGOSACCHARIDE

26974 PECTIN  
 58407 LACTOSE  
 1309 CURDLAN  
 2245 LACTULOSE  
 1544839 BETA  
 16163 GLUCAN  
 8179 BETA(2A)GLUCAN  
 L7 95742 ((ARABINO OR MANNO OR GALACTO OR ISOMALTO OR SIALYL) (W)OLIGOSACC  
 HARIDE) OR ARABINOOLIGOSACCHARIDE OR MANNOOLIGOSACCHARIDE OR  
 GALACTOOLIGOSACCHARIDE OR ISOMALTOOLIGOSACCHARIDE OR SIALYLOLIGO  
 SACCHARIDE OR PECTIN OR LACTOSE OR CURDLAN OR LACTULOSE OR (BETA  
 (2A)GLUCAN)

=> s 15 and 16 and 17

L8 19 L5 AND L6 AND L7

=> file stnguide

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	2.69	67.01
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-15.20

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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.06	67.07
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-15.20

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 FILE LAST UPDATED: 20 Jul 2008 (20080720/ED)

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This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s 18 and (PY<2004 or AY<2004 or PRY<2004)

23986246 PY<2004  
4779965 AY<2004  
4250851 PRY<2004

L9 8 L8 AND (PY<2004 OR AY<2004 OR PRY<2004)

=> file stnguide

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	2.69	69.76
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-15.20

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FILE CONTAINS CURRENT INFORMATION.  
LAST RELOADED: Jul 18, 2008 (20080718/UP).

=> d 19 1-8 ti abs bib  
YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

L9 ANSWER 1 OF 8 HCAPLUS COPYRIGHT 2008 ACS on STN  
TI Oligosaccharide-containing nutritional compositions that inhibit pathogen adhesion to intestinal cells  
AB Saccharides (particularly oligosaccharides) are used as inhibitors of pathogen adhesion to mammalian cells (especially gut cells) and may be used in food and nutritional compns. Compds. are screened for inhibition of adhesion of specific pathogens (verocytotoxic and enteropathogenic Escherichia coli) to the colonic epithelium (HT 29 cell line) without adversely affecting the colonic microflora or adhesion of probiotic organisms. Compds. with suitable activity include mannooligosaccharides, caseinoglycomacropeptides, chitooligosaccharides, galactooligosaccharides, etc.  
AN 2005:283268 HCAPLUS <<LOGINID::20080722>>  
DN 142:335365  
TI Oligosaccharide-containing nutritional compositions that inhibit pathogen adhesion to intestinal cells  
IN Rhoades, Jonathan Robert; Rastall, Robert; Gibson, Glenn R.  
PA Novartis Ag, Switz.  
SO PCT Int. Appl., 31 pp.  
CODEN: PIXXD2  
DT Patent  
LA English

## FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2005027663 WO 2005027663	A2 A3	20050331 20050707	WO 2004-EP10469	20040917 <--
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	BR 2004003979 US 20060287276	A A1	20060221 20061221	BR 2004-3979 US 2006-572664	20040920 <-- 20060320 <--
PRAI	GB 2003-21996 WO 2004-EP10469	A W	20030919 20040917	<--	

L9 ANSWER 2 OF 8 HCAPLUS COPYRIGHT 2008 ACS on STN

TI Prebiotic oligosaccharides: evaluation of biological activities and potential future developments

AB A review. Prebiotics are recognized for their ability to increase levels of 'health promoting' bacteria in the intestinal tract of humans or animals. This normally involves targeting the activities of bifidobacteria and/or lactobacilli. Non digestible oligosaccharides such as fructo-oligosaccharides, lactulose and traps-galacto-oligosaccharides seem to be efficacious prebiotics in that they confer the degree of selective fermentation required. Other oligomers are used as prebiotics in Japan e.g. xylo-oligosaccharides, soybean-oligosaccharides, isomalto-oligosaccharides. To determine prebiotic functionality, various in vitro systems may be used. These range from simple batch culture fermenters to complex models of the gastrointestinal tract. The definitive test however is an in vivo study. The advent of mol. based procedures in gut microbiol. has alleviated many concerns over the reliability of microbial characterization, in response to prebiotic intake. Techniques such as DNA probing and mol. fingerprinting are now being applied to both laboratory and human studies. These will help to further identify prebiotics that can be added to the diet and thereby fortify 'beneficial' bacteria. Such robust technologies can also be used in structure-function assays to identify the mechanisms behind prebiotic effects. Considerable research effort is currently being expended in developing so called 'second generation' prebiotics. These are forms that have multiple biol. activity that attempts health enhancement properties beyond the genus level stimulation of bifidobacteria or lactobacilli within the gut microflora. Examples include higher mol. weight oligomers than is conventional for prebiotics, such that targeted activities in the distal colon are feasible (the left side of the human large gut being the frequent area for colonic disorder). Glycobiol. is also developing anti-adhesive prebiotics that incorporate receptor sites for common gut pathogens and/or their activities. Through the use of reverse enzyme technol., as applied to  $\beta$ -galactosidase activity in prebiotics, oligosaccharides that enhance a lactic microflora at the species, rather than genus, level are possible. This review gives an account of how second generation prebiotics may be manufactured, through a variety of biotechnol. techniques, and tested for their biol. activity. The health attributes of such mols. as well as existing prebiotics is also discussed, with reference to specific target populations. The prebiotic concept is a much more recent development in

dietary intervention for enhanced gut function than is prebiotics. Not surprisingly therefore, research developments are proceeding quickly. Because oligosaccharides can be added to a wide variety of foodstuffs, new functional food developments are continuing. It is important that these are tested using reliable methodologies and that any health effects are underpinned by realistic mechanisms of effect.

AN 2002:783388 HCPLUS <>LOGINID::20080722>>  
DN 138:168911  
TI Prebiotic oligosaccharides: evaluation of biological activities and potential future developments  
AU Rastall, Robert A.; Gibson, Glenn R.  
CS Unit of Food Microbial Sciences, School of Food Biosciences, University of Reading, Reading, RG6 6AP, UK  
SO Probiotics and Prebiotics (2002), 107-148. Editor(s): Tannock, Gerald W. Publisher: Caister Academic Press, Wymondham, UK.  
CODEN: 69DEL7; ISBN: 0-9542464-1-1  
DT Conference; General Review  
LA English  
RE.CNT 99 THERE ARE 99 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 3 OF 8 HCPLUS COPYRIGHT 2008 ACS on STN  
TI The sialylated fraction of milk oligosaccharides is partially responsible for binding to enterotoxigenic and uropathogenic Escherichia coli human strains  
AB Milk oligosaccharides can act as soluble receptors that block bacterial adhesion to the different epithelia. Colonization factor antigens (CFA)/I- and CFA/II-expressing enterotoxigenic Escherichia coli (ETEC) strains constitute one of the main causes of diarrhea in infants. Here, the inhibition of hemagglutination mediated by these strains by milk oligosaccharides was tested. Human milk oligosaccharides showed a strong inhibitory capacity, which decreased when the oligosaccharides were desialylated. Because milk oligosaccharides also are present in the urine of neonates receiving mothers' milk, their ability to bind two uropathogenic Escherichia coli (UPEC) strains was also examined. UPEC strains expressing P (Pap) and P-like (Prs) fimbriae are responsible for infections of the urinary tract such as pyelonephritis and cystitis. The hemagglutination mediated by these strains was inhibited by human milk oligosaccharides. The sialylated fraction was partially responsible for this inhibition in the case of the UPEC expressing the P-like fimbria because differences were found after desialylation. Although bovine milk oligosaccharides were less efficient at inhibiting the hemagglutination of ETEC strains, they were still quite good inhibitors of UPEC strains.

AN 2002:779738 HCPLUS <>LOGINID::20080722>>  
DN 138:24118  
TI The sialylated fraction of milk oligosaccharides is partially responsible for binding to enterotoxigenic and uropathogenic Escherichia coli human strains  
AU Martin-Sosa, Samuel; Martin, Maria-Jesus; Hueso, Pablo  
CS Departamento de Bioquimica y Biologia Molecular, Facultad de Biologia, Universidad de Salamanca, Salamanca, 37007, Spain  
SO Journal of Nutrition (2002), 132(10), 3067-3072  
CODEN: JONUAI; ISSN: 0022-3166  
PB American Society for Nutritional Sciences  
DT Journal  
LA English  
RE.CNT 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 4 OF 8 HCPLUS COPYRIGHT 2008 ACS on STN

TI Blocking adhesion of pathogenic microorganisms to avian cells  
AB For the production of a pharmaceutical preparation for the blocking of adhesion of germs to bird cells one may use oligogalacturonides with a polymerization degree  $\geq 2$  and a degree of esterification < 20% as active substance, optionally together with an ordinary pharmaceutical carrier, auxiliary substances, and fillers into a form suitable for administration to poultry.

AN 2002:446014 HCAPLUS <>LOGINID::20080722>>

DN 137:15762

TI Blocking adhesion of pathogenic microorganisms to avian cells

IN Guggenbichler, Josef Peter; Jurenitsch, Johann

PA de Bettignies-Dutz, Andreas, Germany

SO Ger. Offen., 12 pp.

CODEN: GWXXBX

DT Patent

LA German

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	DE 10061574	A1	20020613	DE 2000-10061574	20001211 <--
	WO 2002047695	A1	20020620	WO 2001-EP14541	20011211 <--
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	AU 2002021947	A5	20020624	AU 2002-21947	20011211 <--
	EP 1341544	A1	20030910	EP 2001-270335	20011211 <--
	EP 1341544	B1	20060524		
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
	AT 326974	T	20060615	AT 2001-270335	20011211 <--
	RU 2281104	C2	20060810	RU 2003-121020	20011211 <--
PRAI	DE 2000-10061574	A	20001211	<--	
	WO 2001-EP14541	W	20011211	<--	

L9 ANSWER 5 OF 8 HCAPLUS COPYRIGHT 2008 ACS on STN

TI Methods for drug administration and distribution based on monitoring blood viscosity and other parameters for diagnostics and treatment

AB Various methods are provided for determining and utilizing the viscosity of the circulating blood of a living being, i.e., a human, over a range of shear rates for diagnostics and treatment, such as detecting/reducing blood viscosity, work of the heart, contractility of the heart, for detecting/reducing the surface tension of the blood, for detecting plasma viscosity, for explaining/countering endothelial cell dysfunction, for providing high and low blood vessel wall shear stress data, red blood cell deformability data, lubricity of blood, and for treating different ailments such as peripheral arterial disease in combination with administering to a living being at least one pharmaceutically acceptable agent. Agents pharmaceutically effective to regulate at least one of the aforementioned blood parameters are used to adjust distribution of a substance through the bloodstream. For example, when blood viscosity is a blood flow parameter monitored, an agent is selected from i.v. diluents, red blood cell deformability agents, antiurea agents, oral contraceptives, antidiabetic agents, antiarrhythmics, antihypertensives, antihyperlipidemics, antiplatelet agents, appetite suppressants, antiobesity agents, blood modifiers, smoking deterrent agents, and

nutritional supplements.  
 AN 2002:185688 HCAPLUS <<LOGINID::20080722>>  
 DN 136:252567  
 TI Methods for drug administration and distribution based on monitoring blood  
 viscosity and other parameters for diagnostics and treatment  
 IN Kensey, Kenneth  
 PA USA  
 SO U.S. Pat. Appl. Publ., 46 pp., Cont.-in-part of U.S. Ser. No. 819,924.  
 CODEN: USXXCO  
 DT Patent  
 LA English  
 FAN.CNT 8

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 20020032149	A1	20020314	US 2001-841389	20010424 <--
	US 6019735	A	20000201	US 1997-919906	19970828 <--
	CA 2301161	A1	19990304	CA 1998-2301161	19980826 <--
	WO 9910724	A2	19990304	WO 1998-US17657	19980826 <--
		W:	AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW		
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	HU 2001000201	A2	20010528	HU 2001-201	19980826 <--
	HU 2001000201	A3	20040329		
	NZ 502905	A	20010831	NZ 1998-502905	19980826 <--
	JP 2001514384	T	20010911	JP 2000-507994	19980826 <--
	US 6322524	B1	20011127	US 1999-439795	19991112 <--
	US 6322525	B1	20011127	US 2000-501856	20000210 <--
	NO 2000000944	A	20000225	NO 2000-944	20000225 <--
	MX 200002073	A	20010821	MX 2000-2073	20000228 <--
	US 6428488	B1	20020806	US 2000-615340	20000712 <--
	WO 2002009583	A2	20020207	WO 2001-US23696	20010730 <--
	WO 2002009583	A3	20020425		
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	US 20020088953	A1	20020711	US 2001-33841	20011227 <--
	US 6624435	B2	20030923		
	WO 2002079778	A2	20021010	WO 2002-US3984	20020207 <--
	WO 2002079778	A3	20030710		
		W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW		
		RW:	GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG		
	US 20020184941	A1	20021212	US 2002-156165	20020528 <--
	US 6571608	B2	20030603		

PRAI	US 1997-919906	A2	19970828	<--
	US 1999-439795	A2	19991112	<--
	US 2000-501856	A2	20000210	<--
	US 2000-628401	A2	20000801	<--
	US 2000-727950	A2	20001201	<--
	US 2001-819924	A2	20010328	<--
	US 1997-966076	A	19971107	<--
	WO 1998-US17657	W	19980826	<--
	US 2000-615340	A3	20000712	<--
	US 2000-228612P	P	20000828	<--
	US 2001-789350	B2	20010221	<--
	US 2001-828761	A	20010409	<--
	US 2001-839785	A	20010420	<--
	US 2001-841389	A	20010424	<--
	US 2001-897164	A3	20010702	<--

L9 ANSWER 6 OF 8 HCPLUS COPYRIGHT 2008 ACS on STN  
 TI Dietary supplement containing histidine for alleviating dysmenorrhea, endometriosis, and pre-term labor  
 AB The present invention relates to methods for alleviating disorders or chronic conditions of the female reproductive system, such as dysmenorrhea, endometrial pain, and pre-term labor, through dietary supplementation with histidine (500 mg-30 g/daily). The invention relates further to novel combination supplements of histidine in conjunction with other nutritional supplement materials which are preferably also useful in alleviating the above-mentioned disorders or conditions. A method for administering a dietary histidine supplement in conjunction with one or more sep. formulated therapeutic drugs also known to be useful in treating these female reproductive conditions is also disclosed. For example, a capsule was prepared containing 300 mg of L-histidine, 250 mg CaCO<sub>3</sub>, and lactose as a carrier.  
 AN 2001:240150 HCPLUS <>LOGINID::20080722>>  
 DN 134:271255  
 TI Dietary supplement containing histidine for alleviating dysmenorrhea, endometriosis, and pre-term labor  
 IN Peterson, Johnny W.; Thomas, Peter G.  
 PA USA  
 SO U.S., 16 pp.  
 CODEN: USXXAM  
 DT Patent  
 LA English  
 FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 6211221	B1	20010403	US 1999-285717	19990405 <--
PRAI US 1999-285717		19990405	<--	

RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD  
 ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 7 OF 8 HCPLUS COPYRIGHT 2008 ACS on STN  
 TI Bright white film coatings and film coating compositions therefor  
 AB A dry film coating composition used to make a bright white film coating for nutritional supplements, pharmaceutical tablets, and the like, comprises dextrose, an auxiliary film-former, and titania. Optionally, but advantageously, the coating composition also may include one or more of the following components: a plasticizer, a surfactant, a flow aid, and a preservative. The composition provides a film coating that possesses good film adhesion and a smooth surface. A coating dispersion was formulated containing dextrose 32, HPMC (Pharmacoat E-50) 10, PEG-8000 8, HPMC (Pharmacoat E-15) 5, Na CMC 6, Na citrate 3, mineral oil 3, titania 32, and Polysorbate-80 1 %. The dispersion was sprayed onto APAP tablets and

AN this produced a bright white film coating.  
DN 1999:77458 HCPLUS <<LOGINID::20080722>>  
DN 130:129995  
TI Bright white film coatings and film coating compositions therefor  
IN Grillo, Susan M.; Korchok, Brian; Kinsey, Bruce; Hartman, Melanie; Porter,  
Stuart C.; Steffenino, Rita; Reyes, George; Burke, Thomas J.  
PA Berwind Pharmaceutical Services, Inc., USA  
SO PCT Int. Appl., 34 pp.  
CODEN: PIXXD2

DT Patent  
LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9903449	A1	19990128	WO 1998-US14830	19980716 <--
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW				
	RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	US 6248391	B1	20010619	US 1997-895484	19970716 <--
	CA 2296425	A1	19990128	CA 1998-2296425	19980716 <--
	CA 2296425	C	20070703		
	AU 9884107	A	19990210	AU 1998-84107	19980716 <--
	AU 738496	B2	20010920		
	EP 1011639	A1	20000628	EP 1998-934621	19980716 <--
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, PT, IE, SI, LT, LV, FI, RO				
	BR 9811106	A	20000718	BR 1998-11106	19980716 <--
	TR 200000122	T2	20000721	TR 2000-122	19980716 <--
	ZA 9806339	A	20001016	ZA 1998-6339	19980716 <--
	JP 2001510149	T	20010731	JP 2000-502751	19980716 <--
	MX 200000570	A	20010731	MX 2000-570	20000114 <--
	US 6267808	B1	20010731	US 2001-754937	20010105 <--
PRAI	US 1997-895484	A	19970716 <--		
	WO 1998-US14830	W	19980716 <--		

RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 8 OF 8 HCPLUS COPYRIGHT 2008 ACS on STN  
TI Invasion of tissue culture cells by diarrheagenic strains of Escherichia  
coli which lack the enteroinvasive inv gene  
AB Invasive Escherichia coli strains of certain serotypes invade by the same  
mechanism as the Shigella sp. It has been proposed that invasion of  
epithelial cells by EPEC strains may also occur; this is a previously  
overlooked property. In the present study E. coli strains isolated from  
patients with diarrhea or ulcerative colitis, lacking the inv  
plasmid mediating classical invasion, but hybridizing with probes for  
different adhesins, were analyzed for their ability to invade HeLa and  
Caco-2 cells. The majority of strains invaded Caco-2 cells to a higher  
extent than HeLa cells. Adhesion to Caco-2 cells was a  
prerequisite for subsequent invasion of the cells but EAF, eae, EAgg and  
other known virulence factors were not sufficient to mediate invasion. In  
8/9 E. coli strains invasion was enhanced after growth under iron  
restriction. Growth during anaerobic conditions did not influence  
subsequent invasion by E. coli strains whereas 6/9 strains had their  
invasive ability significantly decreased after growth in the presence of  
1% glucose. The invasive process was inhibited by mannose but not by

lactose, fucose or galactose. The data indicate that strains of E. coli may invade Caco-2 cells by novel mechanisms which require adhesion to the cells but which differ from those of *Salmonella* sp., *Yersinia* sp., *Shigella* sp. and classical enteroinvasive *E. coli*.  
 AN 1996:347747 HCPLUS <>LOGINID::20080722>>  
 DN 125:83399  
 OREF 125:15695a,15698a  
 TI Invasion of tissue culture cells by diarrheagenic strains of *Escherichia coli* which lack the enteroinvasive inv gene  
 AU Geyid, Aberra; Fletcher, Jon; Gashe, Brehau A.; Ljungh, Asa  
 CS Department of Medical Microbiology, University of Lund, Sörlvegatan 23, Lund, S-223 62, Swed.  
 SO FEMS Immunology and Medical Microbiology (1996), 14(1), 15-24  
 CODEN: FIMIEV; ISSN: 0928-8244  
 PB Elsevier  
 DT Journal  
 LA English

=> d his

(FILE 'HOME' ENTERED AT 13:29:10 ON 22 JUL 2008)  
 FILE 'HCPLUS' ENTERED AT 13:30:24 ON 22 JUL 2008  
 L1 3287 S URSOODEOXYCHOL?  
 L2 139310 S ISCHEM? OR STROKE OR NEUROPROTECTIVE  
 L3 28 S L1 AND L2  
 L4 19 S L3 AND (PY<2005 OR AY<2005 OR PRY<2005)  
 FILE 'STNGUIDE' ENTERED AT 13:30:30 ON 22 JUL 2008  
 FILE 'HCPLUS' ENTERED AT 13:30:43 ON 22 JUL 2008  
 FILE 'STNGUIDE' ENTERED AT 13:30:46 ON 22 JUL 2008  
 FILE 'HCPLUS' ENTERED AT 15:30:51 ON 22 JUL 2008  
 L5 96359 S PREBIOTIC OR ENTERAL OR DIARRHEA OR NUTRITIONAL  
 L6 493913 S ADHESIVE OR ADHESION  
 FILE 'STNGUIDE' ENTERED AT 15:30:54 ON 22 JUL 2008  
 FILE 'HCPLUS' ENTERED AT 15:31:22 ON 22 JUL 2008  
 L7 95742 S ((ARABINO OR MANNO OR GALACTO OR ISOMALTO OR SIALYL) (W)OLIGOS  
 L8 19 S L5 AND L6 AND L7  
 FILE 'STNGUIDE' ENTERED AT 15:31:26 ON 22 JUL 2008  
 FILE 'HCPLUS' ENTERED AT 15:32:11 ON 22 JUL 2008  
 L9 8 S L8 AND (PY<2004 OR AY<2004 OR PRY<2004)  
 FILE 'STNGUIDE' ENTERED AT 15:32:20 ON 22 JUL 2008  
 FILE 'HCPLUS' ENTERED AT 15:32:30 ON 22 JUL 2008  
 FILE 'STNGUIDE' ENTERED AT 15:32:31 ON 22 JUL 2008

=> log hold

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.06	95.85

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-21.60

SESSION WILL BE HELD FOR 120 MINUTES  
 STN INTERNATIONAL SESSION SUSPENDED AT 15:32:35 ON 22 JUL 2008

Connecting via Winsock to STN

Welcome to STN International! Enter x:X

LOGINID:SSPTAEX01623

PASSWORD:

\* \* \* \* \* RECONNECTED TO STN INTERNATIONAL \* \* \* \* \*  
 SESSION RESUMED IN FILE 'STNGUIDE' AT 15:49:38 ON 22 JUL 2008  
 FILE 'STNGUIDE' ENTERED AT 15:49:38 ON 22 JUL 2008  
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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.06	95.85

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-21.60

=> file hcplus

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.12	95.91

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-21.60

FILE 'HCAPLUS' ENTERED AT 15:51:07 ON 22 JUL 2008  
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FILE COVERS 1907 - 22 Jul 2008 VOL 149 ISS 4  
 FILE LAST UPDATED: 21 Jul 2008 (20080721/ED)

HCAplus now includes complete International Patent Classification (IPC) reclassification data for the second quarter of 2008.

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s HT29

L10 2473 HT29

=> s 15 and 16 and l10

L11 1 L5 AND L6 AND L10

=> s 15 and 17 and l10

L12 1 L5 AND L7 AND L10

=> s l11 and (PY<2004 or AY<2004 or PRY<2004)

23986246 PY<2004

4779965 AY<2004

4250851 PRY<2004

L13 0 L11 AND (PY<2004 OR AY<2004 OR PRY<2004)

=> s l12 and (PY<2004 or AY<2004 or PRY<2004)

23986246 PY<2004

4779965 AY<2004

4250851 PRY<2004

L14 0 L12 AND (PY<2004 OR AY<2004 OR PRY<2004)

=> file stnguide

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	2.69	98.60
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-21.60

FILE 'STNGUIDE' ENTERED AT 15:51:16 ON 22 JUL 2008  
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FILE CONTAINS CURRENT INFORMATION.

LAST RELOADED: Jul 18, 2008 (20080718/UP).

=> d l11 ti abs bib

YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

L11 ANSWER 1 OF 1 HCAPLUS COPYRIGHT 2008 ACS on STN  
TI Investigation of the biological activities of pectic oligosaccharides using in vitro models of the human colon  
AB Oligosaccharides derived from pectins by either enzymic hydrolysis or by flash extraction were investigated for their prebiotic activities and for their ability to prevent the adhesion of pathogenic bacteria

and bacterial toxins. Potential prebiotic activity was evaluated using pH-controlled batch cultures inoculated with human faecal samples. Microbial population changes were monitored by fluorescent in situ hybridization techniques. Pectic oligosaccharides derived from either manufacturing route displayed potential prebiotic properties in that they selectively increased the populations of beneficial bacteria such as bifidobacteria and lactobacilli and decreased undesirable bacteria such as clostridia. The oligosaccharides had a more selective fermentation than

the parent polysaccharides. Antiadhesive activity was evaluated using the colon cancer cell line, HT29. Pectic oligosaccharides displayed some degree of protection against Escherichia coli Shiga-like toxins. In addition, pectic oligosaccharides derived by flash extraction from citrus wastes

displayed antiadhesive activity against enteropathogenic and verotoxigenic strains of E. coli. During the execution of this work, we have also discovered preliminary data suggesting that pectic oligosaccharides may act to induce apoptosis in the colon cancer line used.

AN 2005:186972 HCPLUS <<LOGINID::20080722>>

TI Investigation of the biological activities of pectic oligosaccharides using in vitro models of the human colon

AU Rastall, Robert A.; Manderson, Kirstie; Hotchkiss, Arland T.; Gibson, Glenn R.

CS School of Food Biosciences, The University of Reading, Reading, RG6 6AP, UK

SO Abstracts of Papers, 229th ACS National Meeting, San Diego, CA, United States, March 13-17, 2005 (2005), CELL-147 Publisher: American Chemical Society, Washington, D. C.

CODEN: 69GQMP

DT Conference; Meeting Abstract

LA English

=> d 112 ti

YOU HAVE REQUESTED DATA FROM FILE 'HCPLUS' - CONTINUE? (Y)/N:y

L12 ANSWER 1 OF 1 HCPLUS COPYRIGHT 2008 ACS on STN

TI A novel galactooligosaccharide mixture increases the bifidobacterial population numbers in a continuous in vitro fermentation system and in the proximal colonic contents of pigs in vivo

=> d 112 ti abs bib

YOU HAVE REQUESTED DATA FROM FILE 'HCPLUS' - CONTINUE? (Y)/N:y

L12 ANSWER 1 OF 1 HCPLUS COPYRIGHT 2008 ACS on STN

TI A novel galactooligosaccharide mixture increases the bifidobacterial population numbers in a continuous in vitro fermentation system and in the proximal colonic contents of pigs in vivo

AB Prebiotics are nondigestible food ingredients that encourage proliferation of selected groups of the colonic microflora, thereby altering the composition toward a more beneficial community. In the present study, the prebiotic potential of a novel galactooligosaccharide (GOS) mixture, produced by the activity of galactosyltransferases from Bifidobacterium bifidum 41171 on lactose, was assessed in vitro

and in a parallel continuous randomized pig trial. In situ fluorescent hybridization with 16S rRNA-targeted probes was used to investigate changes in total bacteria, bifidobacteria, lactobacilli, bacteroides, and Clostridium histolyticum group in response to supplementing the novel GOS mixture. In a 3-stage continuous culture system, the bifidobacterial nos. for the first 2 vessels, which represented the proximal and traverse colon, increased ( $P < 0.05$ ) after the addition of the oligosaccharide mixture. In addition, the oligosaccharide mixture strongly inhibited the attachment of enterohepatic Escherichia coli ( $P < 0.01$ ) and Salmonella enterica serotype Typhimurium ( $P < 0.01$ ) to HT29 cells. Addition of the novel mixture at 4% (wt:wt) to a com. diet increased the d. of bifidobacteria ( $P < 0.001$ ) and the acetate concentration ( $P < 0.001$ ), and decreased the pH ( $P < 0.001$ ) compared with the control diet and the control diet supplemented with inulin, suggesting a great prebiotic potential for the novel oligosaccharide mixture.

AN 2005:628823 HCAPLUS <>LOGINID::20080722>>  
TI A novel galactooligosaccharide mixture increases the bifidobacterial population numbers in a continuous in vitro fermentation system and in the proximal colonic contents of pigs in vivo  
AU Tzortzis, George; Goulas, Athanasios K.; Gee, Jennifer M.; Gibson, Glenn R.  
CS School of Food Biosciences, The University of Reading, Reading, RG6 6AP, UK  
SO Journal of Nutrition (2005), 135(7), 1726-1731  
CODEN: JONUAI; ISSN: 0022-3166  
PB American Society for Nutritional Sciences  
DT Journal  
LA English  
RE.CNT 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> file hcaplus			
COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION	
FULL ESTIMATED COST	0.18	113.21	
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION	
CA SUBSCRIBER PRICE	0.00	-23.20	

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FILE COVERS 1907 - 22 Jul 2008 VOL 149 ISS 4  
FILE LAST UPDATED: 21 Jul 2008 (20080721/ED)

HCAplus now includes complete International Patent Classification (IPC)

reclassification data for the second quarter of 2008.

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s 15 and l10

L15            26 L5 AND L10

=> s 16 and l10

L16            188 L6 AND L10

=> s 115 and (PY<2004 OR AY<2004 OR PRY<2004)

23986246 PY<2004

4779965 AY<2004

4250851 PRY<2004

L17            15 L15 AND (PY<2004 OR AY<2004 OR PRY<2004)

=> s 116 AND (PY<2004 OR AY<2004 OR PRY<2004)

23986246 PY<2004

4779965 AY<2004

4250851 PRY<2004

L18            115 L16 AND (PY<2004 OR AY<2004 OR PRY<2004)

=> file stnguide

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	2.69	115.90
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-23.20

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FILE CONTAINS CURRENT INFORMATION.

LAST RELOADED: Jul 18, 2008 (20080718/UP).

=> d 117 1-15 ti abs bib

YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

L17 ANSWER 1 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN

TI Method of augmenting the antitumor activity of anticancer agents by selenium compounds

AB A method for augmenting the antitumor activity of anti-cancer agents is provided. The method comprises administering to an individual an anti-cancer agent and a selenium compound. A method is also provided for inhibiting the growth of a tumor which has proven to be refractory to anticancer agents. The methods comprises administration of selenium compound followed by administration of the anticancer agent.

AN 2005:983780 HCAPLUS <<LOGINID::20080722>>  
DN 143:222496  
TI Method of augmenting the antitumor activity of anticancer agents by selenium compounds  
IN Fakih, Marwan; Rustum, Youcef M.; Pendyala, Lakshmi; Smith, Patrick  
PA USA  
SO U.S. Pat. Appl. Publ., 24 pp., Cont.-in-part of U.S. Ser. No. 844,800.  
CODEN: USXXCO

DT Patent  
LA English

FAN.CNT 3

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 20050197399	A1	20050908	US 2005-79633	20050311 <--
	US 20050026852	A1	20050203	US 2004-844800	20040513 <--
	US 20060258697	A1	20061116	US 2006-405377	20060417 <--
PRAI	US 2003-471183P	P	20030513	<--	
	US 2004-844800	A2	20040513		
	US 2005-79633	B2	20050311		

L17 ANSWER 2 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN

TI Serotypes, virulence factors, antibiotic sensitivity, beta-lactamase activity and plasmid analysis of *Salmonella* from children with diarrhea in Tripoli (Libya)

AB A total of 21 *Salmonella* strains isolated in Libya (16 from children with diarrhea and 5 from healthy controls) were serotyped and studied for their cell invasive ability, production of cytotoxin, antibiotic susceptibility,  $\beta$ -lactamase activity and plasmid profiles. Eight different serotypes of *Salmonella* were identified: 6 *S. saintpaul*, 4 *S. wien* (1 from control), 2 *S. newport*, 2 *S. muenchen* (1 from control), 2 *S. typhimurium* (1 from control), 2 *S. hadar* (1 from control), 2 *S. reading* (1 from control), 1 *S. kottbus*. Twenty (95%) were pos. in the invasiveness assay using HeLa cells, and all (100%) were neg. for cytotoxin production in HT29 cells. More than 40% were resistant to ampicillin, cefalexin, cefamandole, cefoperazone, chloramphenicol, gentamicin, mezlocillin and trimethoprim-sulfamethoxazole and 100% were susceptible to the new quinolones. Most (67%) of the strains harbored plasmids and 43% produced  $\beta$ -lactamase. A strong association was observed between the presence of more than one plasmid,  $\beta$ -lactamase activity, and multiple-resistance to antimicrobial agents and serotypes *S. saintpaul* and *S. wien*. Curing expts. with acridine orange showed that 2 plasmids (33 and 1.4 megadaltons) might be responsible for the resistance to chloramphenicol and gentamicin. The present study demonstrated that multiple-resistant salmonellae are widespread in Libya and the resistance is mainly plasmid mediated.

AN 2003:100203 HCAPLUS <<LOGINID::20080722>>  
DN 138:300365

TI Serotypes, virulence factors, antibiotic sensitivity, beta-lactamase activity and plasmid analysis of *Salmonella* from children with diarrhea in Tripoli (Libya)

AU El-Ghodban, A.; Ghenghesh, K. S.; Marialigeti, K.; Abeid, S.

CS Department of Microbiology, Eotvos Lorand University, Budapest, H-1117, Hung.

SO Acta Microbiologica et Immunologica Hungarica (2002), 49(4), 433-444

CODEN: AMIHEF; ISSN: 1217-8950

PB Akademiai Kiado

DT Journal

LA English

RE.CNT 48 THERE ARE 48 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 3 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN

TI Human serum amyloid A3 peptide enhances intestinal MUC3 expression and inhibits EPEC adherence

AB We previously determined that the N-terminal region of bovine mammary-associated

serum amyloid A3 (M-SAA3) increased intestinal mucin MUC3 levels in HT29 human intestinal cells by .apprx.2.5-fold, relative to untreated cells. This study shows that the human M-SAA3 N-terminal peptide further enhances MUC3 transcript levels by .apprx.4.3-fold in these cells ( $p < 0.02$ ), implicating a species-specific interaction. Furthermore, immunofluorescence and immunoblot anal. using a MUC3-specific monoclonal antibody confirms that the human M-SAA3 peptide stimulates MUC3 protein expression and secretion by the HT29 cells. More importantly, pretreatment of the cells with the peptide causes a subsequent 73% decrease in the adherence of enteropathogenic Escherichia coli (EPEC) to these cells, relative to untreated cells ( $p < 0.01$ ). The intestinal mucin MUC3 has been shown to provide a protective barrier in the gut and inhibit adherence of pathogens to the gut wall. Therefore, a means to increase MUC3 protein expression by a colostrum-associated peptide or protein may be a highly effective prophylactic treatment for the prevention of gastrointestinal diseases such as necrotizing enterocolitis and infectious diarrhea.

AN 2002:972020 HCAPLUS <>LOGINID::20080722>>

DN 139:78878

TI Human serum amyloid A3 peptide enhances intestinal MUC3 expression and inhibits EPEC adherence

AU Larson, Marilynn A.; Wei, Shu H.; Weber, Annika; Mack, David R.; McDonald, Thomas L.

CS Department of Pathology and Microbiology, University of Nebraska Medical Center, Omaha, NE, 68198, USA

SO Biochemical and Biophysical Research Communications (2003), 300(2), 531-540

CODEN: BBRCA9; ISSN: 0006-291X

PB Elsevier Science

DT Journal

LA English

RE.CNT 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 4 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN

TI Enteroinvasive bacteria alter barrier and transport properties of human intestinal epithelium: role of iNOS and COX-2

AB Various invasive pathogens cause diarrhea, but the mechanism(s) are poorly understood. We hypothesized that nitric oxide and prostaglandins might modulate chloride secretory and barrier properties of the infected intestinal epithelium and that diarrhea is caused, in part, by altered expression of inducible NO synthase (iNOS) and cyclooxygenase 2 (COX-2). Studies were conducted in human intestinal epithelial cell lines (HT29/cl.19A, Caco-2, and T84). Cells were infected with enteroinvasive Escherichia coli (EIEC 029:NM) or Salmonella dublin (SD), or nonpathogenic, noninvasive bacteria (*Streptococcus thermophilus* [ST] and *Lactobacillus acidophilus* [LA]). Infected cells and controls were tested for transepithelial resistance, chloride secretion, prostaglandin E2, guanosine 3',5'-cyclic monophosphate and adenosine 3',5'-cyclic monophosphate, and protein expression. Cells infected with EIEC or SD, but not uninfected controls or ST/LA-exposed monolayers, showed a progressive reduction in transepithelial resistance starting at 6-12 h. Infected HT29/cl.19A and Caco-2 cells, but not T84 cells, also showed an increase in total nitrite. Expression of iNOS, and consequently COX-2, was also increased, followed by increased

production of prostaglandins and cyclic nucleotides. Furthermore, basal and stimulated chloride secretory responses to various agonists were enhanced in HT29/c1.19A and Caco-2 cells after infection with enteroinvasive bacteria, and this effect was reversed for some agonists by iNOS or COX-2 inhibitors. Increased expression of cystic fibrosis transmembrane conductance regulator and NKCC1 was also observed in EIEC or SD-infected cells vs. controls, secondary to NO synthase activity. Conclusions: Up-regulation of iNOS and COX-2 by enteroinvasive bacteria can modulate chloride secretion and barrier function in intestinal epithelial cells. Thus, these enzymes represent possible therapeutic targets in infectious diarrhea.

AN 2002:317594 HCPLUS <>LOGINID::20080722>>

DN 137:199457

TI Enteroinvasive bacteria alter barrier and transport properties of human intestinal epithelium: role of iNOS and COX-2

AU Resta-Lenert, Silvia; Barrett, Kim E.

CS Department of Medicine, University of California, San Diego, School of Medicine, San Diego, CA, USA

SO Gastroenterology (2002), 122(4), 1070-1087  
CODEN: GASTAB; ISSN: 0016-5085

PB W. B. Saunders Co.

DT Journal

LA English

RE.CNT 56 THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 5 OF 15 HCPLUS COPYRIGHT 2008 ACS on STN

TI A functional NSP4 enterotoxin peptide secreted from rotavirus-infected cells

AB Previous studies have shown that the nonstructural glycoprotein NSP4 plays a role in rotavirus pathogenesis by functioning as an enterotoxin. One prediction of the mechanism of action of this enterotoxin was that it is secreted from virus-infected cells. In this study, the media of cultured (i) insect cells infected with a recombinant baculovirus expressing NSP4, (ii) monkey kidney (MA104) cells infected with the simian (SA11) or porcine attenuated (OSU-a) rotavirus, and (iii) human intestinal (HT29) cells infected with SA11 were examined to determine if NSP4 was detectable. Sodium dodecyl sulfate-PAGE-Western blotting, immunopptn. and N-terminal amino acid sequencing identified, in the early media from virus-infected cells, a secreted, cleavage product of NSP4 with an apparent mol. weight of 7,000 that represented amino acids 112 to 175 (NSP4 aa112-175). The secretion of NSP4 aa112-175 was not affected by treatment of cells with brefeldin A but was abolished by treatment with nocodazole and cytochalasin D, indicating that secretion of this protein occurs via a nonclassical, Golgi apparatus-independent mechanism that utilizes the microtubule and actin microfilament network. A partial gene fragment coding for NSP4 aa112-175 was cloned and expressed using the baculovirus-insect cell system. Purified NSP4 aa112-175 increased intracellular calcium mobilization in intestinal cells when added exogenously, and in insect cells when expressed endogenously, similar to full-length NSP4. NSP4 aa112-175 caused diarrhea in neonatal mice, as did full-length NSP4. These results indicate that NSP4 aa112-175 is a functional NSP4 enterotoxin peptide secreted from rotavirus-infected cells.

AN 2001:205105 HCPLUS <>LOGINID::20080722>>

DN 135:3857

TI A functional NSP4 enterotoxin peptide secreted from rotavirus-infected cells

AU Zhang, Mingdong; Zeng, Carl Q.-Y.; Morris, Andrew P.; Estes, Mark K.

CS Division of Molecular Virology, Baylor College of Medicine, Houston, TX, 77030, USA

SO Journal of Virology (2000), 74(24), 11663-11670  
CODEN: JOVIAM; ISSN: 0022-538X

PB American Society for Microbiology  
DT Journal  
LA English

RE.CNT 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 6 OF 15 HCPLUS COPYRIGHT 2008 ACS on STN

TI Inter- and intra-individual variation of fecal water - genotoxicity in  
human colon cells

AB Exogenous nutritional factors modulate the fecal contents leading to an enhanced or reduced burden with toxic and cancerogenic factors. These factors are thought to contribute to colon cancer by inducing mutations or enhancing proliferation in colon cells. Fecal water more or less causes these effects in model systems and thus could be the basis for valuable biomarker approaches. Our investigations are aimed at determining geno- and cytotoxicity of fecal water in human colon cell lines in vitro. We are developing techniques for their applicability as biomarker tests during dietary intervention studies. Fecal water is isolated by centrifugation of the feces at 25 000+g and added to cultured human colon cells (HT29). Membrane damage as assessed by trypan blue exclusion is determined as a measure for cytotoxicity. Semiquant. anal. of inducible DNA damage (breaks and alkali labile sites) are analyzed with the single cell microgel electrophoresis assay (comet-assay) and oxidized DNA bases by the addnl. use of repair specific enzymes. We have now determined baseline toxic activities and calculated inter- and intra-individual and -exptl. coeffs. of variation for fecal water from different subjects consuming similar or different diets. Most fecal water induced DNA damage and oxidized DNA bases in HT29 clone 19a cells (0.9-9.14 fold and 1.7-4.9 fold, resp. in comparison to the NaCl controls). Intra- and inter-exptl. coeffs. (CV) of variation, were in a similar order of magnitude and ranged from 6.9 to 31.4. In contrast both intra- and inter-individual variability were considerably higher (CV-ranges of 29.7-76.6 and 21.3-64.0, resp.). Interestingly, these inter-individual values were not lowered when subjects consumed identical diets (CV-ranges of 28.4-126.0). However, following intervention with certain protective dietary regimens (e.g. lignan containing bread) significant redns. of fecal water-induced genotoxicity can be observed. Therefore, in spite of the expected and observed degrees of variation in this methodol., effective exptl. protocols may still lead to detectable modulations of the level of toxic and genotoxic effects.

AN 2000:875450 HCPLUS <<LOGINID::20080722>>

DN 134:158721

TI Inter- and intra-individual variation of fecal water - genotoxicity in  
human colon cells

AU Osswald, K.; Becker, T. W.; Grimm, M.; Jahreis, G.; Pool-Zobel, B. L.  
CS Institute for Nutrition, Department of Nutritional Toxicology,

Friedrich-Schiller University, Jena, D-07743, Germany

SO Mutation Research, Genetic Toxicology and Environmental Mutagenesis (2000), 472(1-2), 59-70  
CODEN: MRGMFI; ISSN: 1383-5718

PB Elsevier B.V.

DT Journal

LA English

RE.CNT 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 7 OF 15 HCPLUS COPYRIGHT 2008 ACS on STN

TI Application of confocal laser scanning microscopy to detect oxidative stress in human colon cells

AB Introduction - Excess of intracellular reactive oxygen species in relation to antioxidant systems results in an oxidative environment which may modulate gene expression or damage cellular mols. These events are expected to greatly contribute to processes of carcinogenesis. Only few studies are available on the oxidative/reductive conditions in the colon, an important tumor target tissue. It was the objective of this work to further develop methods to assess intracellular oxidative stress within human colon cells as a tool to study such assocns. in nutritional toxicol. Methods - We have measured H<sub>2</sub>O<sub>2</sub>-induced oxidative stress in different colon cell lines, in freshly isolated human colon crypts, and, for comparative purposes, in NIH3T3 mouse embryo fibroblasts. Detection was performed by loading the cells with the fluorogenic peroxide-sensitive dye 6-carboxy-2',7'-dichlorodihydrofluorescein diacetate (diacetoxymethyl ester), followed by in vitro treatment with H<sub>2</sub>O<sub>2</sub> and fluorescence detection with confocal laser scanning microscopy (CLSM). Using the microgel electrophoresis ("Comet") Assay, we also examined HT29 stem and clone 19A cells and freshly isolated primary colon cells for their relative sensitivity toward H<sub>2</sub>O<sub>2</sub>-induced DNA damage and for steady-state levels of endogenous oxidative DNA damage. Results A dose-response relationship was found for the H<sub>2</sub>O<sub>2</sub>-induced dye decomposition in NIH3T3 cells (7.8-125 μM H<sub>2</sub>O<sub>2</sub>) whereas no effect occurred in the human colon tumor cell lines HT29 stem and HT29 clone 19A (62-1000 μM H<sub>2</sub>O<sub>2</sub>). Fluorescence was significantly increased at 62 μM H<sub>2</sub>O<sub>2</sub> in the human colon adenocarcinoma cell line Caco-2. In isolated human colon crypts, the lower crypt cells (targets of colon cancer) were more sensitive towards H<sub>2</sub>O<sub>2</sub> than the more differentiated upper crypt cells. In contrast to the CLSM results, oxidative DNA damage was detected in both cell lines using the Comet Assay. Endogenous oxidative DNA damage was highest in HT29 clone 19A, followed by the primary colon cells and HT29 stem cells. Conclusions Oxidative stress in colon cells leads to damage of macromols. which is sensitively detected in the Comet Assay. The lacking response of the CLSM-approach in colon tumor cells is probably due to intrinsic modes of protective activities of these cells. In general, however, the CLSM method is a sensitive technique to detect very low concns. of H<sub>2</sub>O<sub>2</sub>-induced oxidative stress in NIH3T3 cells. Moreover, by using colon crypts it provides the unique possibility of assessing cell specific levels of oxidative stress in explanted human tissues. Our results demonstrate that the actual target cells of colon cancer induction are indeed susceptible to the oxidative activity of H<sub>2</sub>O<sub>2</sub>.

AN 2000:763779 HCPLUS <>LOGINID::20080722>>

DN 134:53301

TI Application of confocal laser scanning microscopy to detect oxidative stress in human colon cells

AU Liegibel, Ute M.; Abrahamse, Salomon L.; Pool-Zobel, Beatrice L.; Rechkemmer, Gerhard

CS Department of Nutritional Toxicology, Institute for Nutrition, Friedrich-Schiller-University, Jena, 07743, Germany

SO Free Radical Research (2000), 32(6), 535-547

CODEN: FRARER; ISSN: 1071-5762

PB Harwood Academic Publishers

DT Journal

LA English

RE.CNT 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 8 OF 15 HCPLUS COPYRIGHT 2008 ACS on STN

TI The in vitro manipulation of carbohydrate metabolism: a new strategy for deciphering the cellular defence mechanisms against nitric oxide attack

AB This study was aimed at examining the effects of manipulating the carbohydrate source of the culture medium on the cellular sensitivity of

epithelial cells to an oxidative attack. Our rationale was that substituting galactose for glucose in culture media would remove the protection afforded by glucose utilization in two major metabolic pathways, i.e. anaerobic glycolysis and/or the pentose phosphate pathway (PPP), which builds up cellular reducing power. Indeed, we show that the polarized human colonic epithelial cell line HT29-C1.16E was sensitive to the deleterious effects of the NO donor PAPANONOate [3-(2-hydroxy-2-nitroso-1-propylhydrazino)-1-propanamine] only in galactose-containing medium. In such medium NO attack led to cytotoxic and apoptotic cell death, associated with formation of derivs. of NO auto-oxidation (collectively termed NO<sub>x</sub>) and peroxynitrite, leading to intracellular GSH depletion and nitrotyrosine formation. The addition of 2-deoxyglucose, a non-glycolytic substrate, to galactose-fed cells protected HT29-C1.16E cells from NO attack and maintained control GSH levels through its metabolic utilization in the PPP, as shown by <sup>14</sup>CO<sub>2</sub> production from 2-deoxy[1-<sup>14</sup>C]glucose. Therefore, increasing the availability of reducing equivalent without interfering with energy metabolism is able to prevent NO-induced cell injury. Finally, this background provides the conceptual framework for establishing nutritional manipulation of cellular metabolic pathways that could provide new means for (i) deciphering the mechanisms of cell injury by reactive nitrogen species and reactive oxygen species at the whole-cell level and (ii) establishing the hierarchy of intracellular defense mechanisms against these attacks.

AN 2000:27432 HCAPLUS <<LOGINID::20080722>>

DN 132:178542

TI The in vitro manipulation of carbohydrate metabolism: a new strategy for deciphering the cellular defence mechanisms against nitric oxide attack

AU Le Goffe, Claire; Vallette, Genevieve; Jarry, Anne; Bou-Hanna, Chantal; Laboisse, Christian L.

CS INSERM CJF 94-04, Faculte de Medecine, Nantes, 44035, Fr.

SO Biochemical Journal (1999), 344(3), 643-648

CODEN: BIJOAK; ISSN: 0264-6021

PB Portland Press Ltd.

DT Journal

LA English

RE.CNT 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 9 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN

TI Rapid and sensitive assay for detection of enterotoxigenic *Bacteroides fragilis*

AB *Bacteroides fragilis* is an obligatory anaerobic, gram-neg. bacterium found among the normal intestinal flora of humans. Enterotoxigenic strains of *B. fragilis* (ETBF) have been associated with diarrheal diseases in humans and animals. The enterotoxin of ETBF induces fluid changes in ligated intestinal segments and cytotoxic response in HT29/C1 cells. By using a pair of monoclonal antibodies (MAbs; MAb C3 and MAb 4H8) specific for the lipopolysaccharide of *B. fragilis*, an assay based on immunomagnetic separation (IMS) in combination with PCR (IMS-PCR) was developed. After DNA extraction, a 294-bp fragment was amplified. The specificity of the IMS-PCR assay was evaluated by adding previously isolated and confirmed ETBF strains to normal fecal samples. All fecal samples to which ETBF strains were added were pos., showing a 100% specificity. The spiked fecal samples were also used for evaluation of the sensitivity of the assay. The detection limit was found to be .apprx.50 CFU/g of feces. By this method 10 clin. fecal samples (5 from patients with diarrhea and 5 from healthy controls) were examined. The results of PCR were in accordance with the results of the HT29/C1 cell assay for all samples. The min. time to retrieval of the final result by the IMS-PCR method is 36 h. The proposed IMS-PCR assay is rapid and sensitive for the direct detection of ETBF in stool samples.

AN 1998:787491 HCAPLUS <<LOGINID::20080722>>  
DN 130:179434  
TI Rapid and sensitive assay for detection of enterotoxigenic *Bacteroides fragilis*  
AU Zhang, Guangming; Weintraub, Andrej  
CS Department of Immunology, Microbiology, Pathology and Infectious Diseases,  
Division of Clinical and Oral Bacteriology, Karolinska Institute, Huddinge  
University Hospital, Huddinge, S-141 86, Swed.  
SO Journal of Clinical Microbiology (1998), 36(12), 3545-3548  
CODEN: JCMIDW; ISSN: 0095-1137  
PB American Society for Microbiology  
DT Journal  
LA English

RE.CNT 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 10 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN  
TI Osmotic changes and ethanol modify TFF gene expression in gastrointestinal cell lines  
AB The gastrointestinal tract is exposed to environmental insult as a result of food intake or in pathol. conditions such as diarrhea, and is therefore protected by the mucus layer. As part of it, trefoil factor family peptides (TFFs) are able to modify the viscoelastic properties of the mucus, protect against exptl. ulceration, and promote repair of the epithelia. The authors investigated, using transient reporter gene assays and RT-PCR in the gastric carcinoma cell line MKN45 and colon carcinoma cell lines LS174T and HT29, whether ethanol and osmotic changes can modify transcriptional activity of TFFs. In a mild hypotonic environment (200 mosmol/L) all three TFF genes were up-regulated by at least a factor of 2. In hypertonic medium (400 mosmol/l), TFF1 and TFF3 were down-regulated, whereas TFF2 was up-regulated by elevated concns. of sodium or chloride in MKN45. Raising the osmolality by ethanol resulted in an up-regulation of TFF3 in both colon cell lines but not in the gastric cell line. The authors conclude that alteration in TFF gene expression is a response of gut epithelia to deal with osmotic forces and ethanol.

AN 1998:766907 HCAPLUS <<LOGINID::20080722>>  
DN 130:91534  
TI Osmotic changes and ethanol modify TFF gene expression in gastrointestinal cell lines  
AU Ludeking, Alexander; Fegert, Petra; Blin, Nikolaus; Gott, Peter  
CS Department of Anthropology and Human Genetics, Division of Molecular Genetics, University of Tubingen, Tubingen, D-72074, Germany  
SO FEBS Letters (1998), 439(1,2), 180-184  
CODEN: FEBLAL; ISSN: 0014-5793  
PB Elsevier Science B.V.  
DT Journal  
LA English

RE.CNT 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 11 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN  
TI In vitro inhibition of *Cryptosporidium parvum* infection by human monoclonal antibodies  
AB *Cryptosporidium parvum* infection of the small epithelial intestine causes unremitting diarrhea and malabsorption that can lead to chronic and sometimes fatal illness in patients with AIDS. The illness may be ameliorated by passive oral Ig therapy. The objective of this study was to produce anti-*Cryptosporidium* human monoclonal antibodies for evaluation as potential therapy. All human monoclonal cell lines that produced *C. parvum* antibodies were originally generated from the peripheral blood

lymphocytes of a human immunodeficiency virus-seroneg. woman. She had recovered from *C. parvum* infection and had a high specific antibody titer. Hybridization of these lymphocytes with a tumor cell line was accomplished by hypo-osmolar electrofusion. Twelve clones were identified by ELISA as secreting anti-*Cryptosporidium* antibodies after the initial hybridization. From the 12 pos. clones, two high antibody-secreting clones, 17A and 17B, were maintained in long-term culture. A second hybridization produced two other human monoclonal cell lines, EC5 and BB2. Human monoclonal antibody from the first two cell lines bound to *C. parvum* sporozoites and oocysts by immunofluorescence. The ability of human monoclonal antibodies to inhibit *C. parvum* infection in vitro was assessed by using a human enterocyte cell line, HT29.74. The antibodies of the four different human hybridomas inhibited infection by 35 to 68% compared to a control irrelevant human monoclonal antibody derived in a similar fashion. Human monoclonal antibodies are candidate mols. for immunotherapy of *C. parvum* infection.

AN 1997:596458 HCPLUS <<LOGINID::20080722>>  
DN 127:277063  
OREF 127:54105a,54108a  
TI In vitro inhibition of *Cryptosporidium parvum* infection by human monoclonal antibodies  
AU Elliot, Bethany C.; Wisnewski, Adam V.; Johnson, Joan; Fenwick-Smith, Daniela; Wiest, Peter; Hamer, David; Kresina, Thomas; Flanigan, Timothy P.  
CS Miriam Hospital, Brown University, Providence, RI, 02906, USA  
SO Infection and Immunity (1997), 65(9), 3933-3935  
CODEN: INFIBR; ISSN: 0019-9567  
PB American Society for Microbiology  
DT Journal  
LA English  
RE.CNT 16 THERE ARE 16 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 12 OF 15 HCPLUS COPYRIGHT 2008 ACS on STN  
TI Bacteroides fragilis toxin rearranges the actin cytoskeleton of HT29/C1 cells without direct proteolysis of actin or decrease in F-actin content  
AB Enterotoxigenic strains of *B. fragilis* associated with childhood diarrhea produce a 20 kDa zinc metalloprotease toxin (BFT). BFT is reported to cleave G-actin in vitro and also causes dramatic rounding and rearrangement of the F-actin cytoskeleton in human intestinal epithelial cell lines (HT29 and HT29/C1). To test the hypothesis that the proteolysis of cellular actin by BFT in vivo may contribute to these alterations in morphol. and cytoskeletal architecture, the authors assessed the F-actin content and the arrangement of the F- and G-actin cytoskeleton in BFT-treated HT29/C1 cells by spectrofluorimetry, confocal microscopy, and immunoblotting. BFT-treated cells were compared to cells treated with *C. difficile* toxin A (CDA) or cytochalasin D. Using spectrofluorometric quantification, the F-actin content of BFT- and cytochalasin D-treated cells was unchanged in contrast to a significant decrease in CDA-treated cells. By confocal microscopy, the arrangement of F- and G-actin in all treated cells was markedly different than control cells. There was no change in the immunoblotting pattern of actin in the Triton-soluble or -insol. cellular fractions of BFT-treated HT29/C1 cells. Evidently, BFT alters the F- and G-actin cytoskeletal architecture of HT29/C1 cells without direct proteolysis of actin or decrease in F-actin content.

AN 1997:408968 HCPLUS <<LOGINID::20080722>>  
DN 127:46295  
OREF 127:8727a,8730a  
TI Bacteroides fragilis toxin rearranges the actin cytoskeleton of HT29/C1 cells without direct proteolysis of actin or decrease in

F-actin content  
AU Saidi, Roxan F.; Jaeger, Kristin; Montrose, Marshall H.; Wu, Shaoguang;  
Sears, Cynthia L.  
CS Division of Gastroenterology, Department of Medicine, Johns Hopkins  
University School of Medicine, Baltimore, MD, 21205, USA  
SO Cell Motility and the Cytoskeleton (1997), 37(2), 159-165  
CODEN: CMCYEO; ISSN: 0886-1544  
PB Wiley-Liss  
DT Journal  
LA English

L17 ANSWER 13 OF 15 HCPLUS COPYRIGHT 2008 ACS on STN  
TI Bacteroides fragilis toxin rapidly intoxicates human intestinal epithelial  
cells (HT29/C1) in vitro  
AB Enterotoxigenic Bacteroides fragilis strains associated with childhood  
diarrhea produce a 20-kDa protein toxin (BFT). Purified BFT  
causes striking morphol. changes in subconfluent human colonic epithelial  
cells (HT29/C1). In a 3-h HT29/C1 cell assay, the  
estimated half-maximal effective concentration of BFT was 12.5 pM, and morphol.  
effects were detectable as early as 30 min and nearly complete by 1.5 h.  
Concns. as low as 0.5 pM could also cause intoxication, but morphol.  
changes were detectable only when the assay was extended to 18 h. The  
onset of this intoxication was concentration dependent and rapid, occurring  
within minutes (<7 min at 0.25 nM, <2 min at 2.5 nM). Notably, the onset  
of intoxication at 37° became irreversible to washing within 2 min  
after exposure to BFT. Morphol. changes were completely inhibited by  
treatment of HT29/C1 cells with BFT at 4° but could be  
demonstrated by subsequent warming to temps. of 15° or higher after  
washing. The time required for the association of BFT with HT29/C1  
cells at 4° but could be demonstrated by subsequent warming to  
temps. of 15° or higher after washing. The time required for the  
association of BFT with HT29/C1 4° was inversely correlated  
with concentration. Inhibitors of endosomal and Golgi trafficking (NH4Cl and  
brefeldin A) prevented the intoxication of HT29/C1 cells by  
Clostridium difficile toxin A and cholera toxin, resp., but not by BFT.  
Agents altering microtubule structure did not affect the cellular activity  
of BFT. These data indicate that a purified toxin from B. fragilis  
strains associated with diarrhea rapidly and irreversibly  
intoxicates human intestinal epithelial cells (HT29/C1) in a  
concentration- and temperature-dependent manner and that the process of  
intoxication  
may not involve internalization mechanisms utilizing microtubules or  
sensitive to pH or brefeldin A.  
AN 1996:718615 HCPLUS <>LOGINID::20080722>>  
DN 126:15745  
OREF 126:3225a,3228a  
TI Bacteroides fragilis toxin rapidly intoxicates human intestinal epithelial  
cells (HT29/C1) in vitro  
AU Saidi, Roxan F.; Sears, Cynthia L.  
CS Div. Gastroenterol. Infectious Diseases, Johns Hopkins Univ. Sch. Med.,  
Baltimore, MD, 21205, USA  
SO Infection and Immunity (1996), 64(12), 5029-5034  
CODEN: INFIBR; ISSN: 0019-9567  
PB American Society for Microbiology  
DT Journal  
LA English  
RE.CNT 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 14 OF 15 HCPLUS COPYRIGHT 2008 ACS on STN  
TI Human intestinal epithelial cells swell and demonstrate actin

- rearrangement in response to the metalloprotease toxin of *Bacteroides fragilis*
- AB Enterotoxigenic *Bacteroides fragilis* (ETBF) cells produce a 20-kDa heat-labile metalloprotease toxin which is potentially important in the pathogenesis of diarrhea associated with this infection. Previous studies indicate that subconfluent HT29/C1 cells treated with the *B. fragilis* toxin (BFT) develop morphol. changes with dissoln. of tight clusters and apparent swelling. Such alterations suggest toxin-stimulated reorganization of the cellular cytoskeleton. The purpose of the current study was to evaluate the effect of *B. fragilis* toxin (BFT) on actin microfilaments (F-actin) and cell volume As assessed by fluorescent phalloidin staining which detects F-actin, BFT treatment of HT29/C1 cells resulted in redistribution of F-actin with loss of stress fibers, a floccular staining pattern, and cellular membrane blebbing without quant. changes in F-actin fluorescence intensity. The F-actin redistribution was time and concentration dependent. In contrast to the cell shrinkage observed in response to the F-actin-depolymg. agents cytochalasin D and *Clostridium difficile* toxin A, BFT stimulated an increase in HT29/C1 cell volume of 10 to 25% (compared with control cells) over a 24-h time course. Only 10 to 30 ng of BFT per mL was necessary to stimulate a maximal increase in HT29/C1 cell volume The effect of BFT on cell volume was persistent and dependent on the proteolytic activity of BFT. In agreement with cell viability assays indicating that BFT did not injure HT29/C1 cells, intoxicated cells exhibited regulatory volume decrease, suggesting that toxin-treated cells remain physiol. dynamic. We conclude that BFT acts on the intestinal epithelial cell cytoskeleton to alter F-actin structure and to stimulate an increase in HT29/C1 cell volume Although these two activities of BFT appear to be linked, the precise sequence of cellular events following intoxication of HT29/C1 cells with BFT remains unclear. We hypothesize that these F-actin and cell volume changes may lead to an alteration in tight junction function in the polarized intestinal epithelium, contributing to the pathogenesis of diarrhea in ETBF infections.
- AN 1996:718610 HCAPLUS <<LOGINID::20080722>>
- DN 126:15744
- OREF 126:3225a,3228a
- TI Human intestinal epithelial cells swell and demonstrate actin rearrangement in response to the metalloprotease toxin of *Bacteroides fragilis*
- AU Koshy, Sherin S.; Montrose, Marshall H.; Sears, Cynthia L.
- CS Div. Infectious diseases Gastroenterol., Johns Hopkins Univ. Sch. Med., Baltimore, MD, 21205-2196, USA
- SO Infection and Immunity (1996), 64(12), 5022-5028
- CODEN: INFIBR; ISSN: 0019-9567
- PB American Society for Microbiology
- DT Journal
- LA English
- RE.CNT 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT
- L17 ANSWER 15 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
- TI Human colon epithelial cells productively infected with human immunodeficiency virus show impaired differentiation and altered secretion
- AB Selected strains of the human immunodeficiency virus (HIV) types 1 and 2 are able to infect human colon epithelial cells in vitro, suggesting a mechanism for the anal route of HIV transmission. In some cases, HIV is not produced by infected colon cells but can be rescued after coculture with T-lymphoid cells. One of the HIV strains (HIV1-NDK) replicated well in colonic cells. A transmission electron microscope study demonstrated

two major structural perturbations in producer colon cells: an unusual number of secretion bodies and the appearance of intracellular lumina with disorganized microvilli, indicating a defect in brush border assembly and differentiation. Either abnormality could account for HIV-induced enteropathy consisting of chronic diarrhea and malabsorption in the absence of enteric pathogens. Moreover, HT29 cells infected with HIV provide a unique model for selection of enterotropic HIV strains.

AN 1992:57208 HCPLUS <>LOGINID::20080722>>  
DN 116:57208  
OREF 116:9895a,9898a  
TI Human colon epithelial cells productively infected with human immunodeficiency virus show impaired differentiation and altered secretion  
AU Fantini, Jacques; Yahi, Nouara; Baghdiguian, Stephen; Chermann, Jean Claude  
CS Univ. Aix-Marseille I, Marseille, 13331, Fr.  
SO Journal of Virology (1992), 66(1), 580-5  
CODEN: JOVIAM; ISSN: 0022-538X  
DT Journal  
LA English

=> d his

(FILE 'HOME' ENTERED AT 13:29:10 ON 22 JUL 2008)

FILE 'HCPLUS' ENTERED AT 13:30:24 ON 22 JUL 2008  
L1 3287 S URSOODEOXYCHOL?  
L2 139310 S ISCHEM? OR STROKE OR NEUROPROTECTIVE  
L3 28 S L1 AND L2  
L4 19 S L3 AND (PY<2005 OR AY<2005 OR PRY<2005)

FILE 'STNGUIDE' ENTERED AT 13:30:30 ON 22 JUL 2008

FILE 'HCPLUS' ENTERED AT 13:30:43 ON 22 JUL 2008

FILE 'STNGUIDE' ENTERED AT 13:30:46 ON 22 JUL 2008

FILE 'HCPLUS' ENTERED AT 15:30:51 ON 22 JUL 2008  
L5 96359 S PREBIOTIC OR ENTERAL OR DIARRHEA OR NUTRITIONAL  
L6 493913 S ADHESIVE OR ADHESION

FILE 'STNGUIDE' ENTERED AT 15:30:54 ON 22 JUL 2008

FILE 'HCPLUS' ENTERED AT 15:31:22 ON 22 JUL 2008  
L7 95742 S ((ARABINO OR MANNO OR GALACTO OR ISOMALTO OR SIALYL) (W)OLIGOS  
L8 19 S L5 AND L6 AND L7

FILE 'STNGUIDE' ENTERED AT 15:31:26 ON 22 JUL 2008

FILE 'HCPLUS' ENTERED AT 15:32:11 ON 22 JUL 2008  
L9 8 S L8 AND (PY<2004 OR AY<2004 OR PRY<2004)

FILE 'STNGUIDE' ENTERED AT 15:32:20 ON 22 JUL 2008

FILE 'HCPLUS' ENTERED AT 15:32:30 ON 22 JUL 2008

FILE 'STNGUIDE' ENTERED AT 15:32:31 ON 22 JUL 2008

FILE 'HCPLUS' ENTERED AT 15:51:07 ON 22 JUL 2008  
L10 2473 S HT29  
L11 1 S L5 AND L6 AND L10

L12           1 S L5 AND L7 AND L10  
L13           0 S L11 AND (PY<2004 OR AY<2004 OR PRY<2004)  
L14           0 S L12 AND (PY<2004 OR AY<2004 OR PRY<2004)

FILE 'STNGUIDE' ENTERED AT 15:51:16 ON 22 JUL 2008

FILE 'HCAPLUS' ENTERED AT 15:51:30 ON 22 JUL 2008

FILE 'STNGUIDE' ENTERED AT 15:51:30 ON 22 JUL 2008

FILE 'HCAPLUS' ENTERED AT 15:51:33 ON 22 JUL 2008

FILE 'STNGUIDE' ENTERED AT 15:51:33 ON 22 JUL 2008

FILE 'HCAPLUS' ENTERED AT 15:51:41 ON 22 JUL 2008

FILE 'STNGUIDE' ENTERED AT 15:51:41 ON 22 JUL 2008

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L15           26 S L5 AND L10  
L16           188 S L6 AND L10  
L17           15 S L15 AND (PY<2004 OR AY<2004 OR PRY<2004)  
L18           115 S L16 AND (PY<2004 OR AY<2004 OR PRY<2004)

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FILE 'HCAPLUS' ENTERED AT 15:53:39 ON 22 JUL 2008

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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.06	162.36
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```
=> exp mannobiose/cn
E1      1      MANNOBIOSE/CN
E2      1      MANNOBIOHYDROLASE, EXO-1,4-B-/CN
E3      1 --> MANNOBIOSE/CN
E4      1      MANNOBIOSE, 1,6-A-/CN
E5      1      MANNODEXTRIN/CN
E6      1      MANNOFRUCTOKINASE/CN
E7      1      MANNOFUCOGALACTAN/CN
E8      1      MANNOFURANOSE, 1,1'-DITHIOBIS(1-DEOXY-2,3:5,6-DI-O-ISOPROPYL
IDENE-, A-D-/CN
E9      1      MANNOFURANOSE, 1,2:5,6-DI-O-ISOPROPYLIDENE-, B-D-/CN
E10     1      MANNOFURANOSE, 1,2:5,6-DI-O-ISOPROPYLIDENE-, ACETATE, B
-D-/CN
E11     1      MANNOFURANOSE, 1,5,6-TRIACETATE CYCLIC 2,3-CARBONATE/CN
E12     1      MANNOFURANOSE, 1,5,6-TRIACETATE CYCLIC 2,3-CARBONATE, .ALPHA
.-D-/CN
```

```
=> s E4
L19      1 "MANNOBIOSE, 1,6-A-"/CN
```

```
=> exp alpha mannobiose/cn
E1      4      ALPHA KG DEPENDENT 2,4-D DIOXYGENASE (BURKHOLDERIA XENOVORAN
S STRAIN LB400)/CN
```

E2 1 ALPHA LIPID 300/CN  
 E3 0 --> ALPHA MANNOBIOSE/CN  
 E4 1 ALPHA MANNOSIDASE (SYNECHOCOCCUS STRAIN WH8102 GENE SYNW0267 )/CN  
 E5 1 ALPHA MANNOSIDASE 6A8B (HUMAN GENE 6A8B)/CN  
 E6 1 ALPHA MANNOSIDASE II ISOZYME (HUMAN CELL LINE SK-MEL-28 CLON E PMX6)/CN  
 E7 1 ALPHA MANNOSIDASE II ISOZYME (HUMAN CELL LINE SK-MEL-28)/CN  
 E8 1 ALPHA MATING PHEROMONE (SACCHAROMYCES NAGANISHII GENE MFALPH A1 PRECURSOR)/CN  
 E9 1 ALPHA MEDOPA/CN  
 E10 1 ALPHA METALS 171/CN  
 E11 1 ALPHA MS/CN  
 E12 1 ALPHA NAC (ARABIDOPSIS THALIANA GENE F7L13.60)/CN

=> exp alpha 1,3-mannobiose/cn

E1 2 ALPHA 1,3-FUCOSYLTRANSFERASE (HELICOBACTER PYLORI STRAIN HPA G1)/CN  
 E2 1 ALPHA 1,3-FUCOSYLTRANSFERASE FUC-T (SIMILAR TO MOUSE FUT4) ( RATTUS NORVEGICUS CLONE MGC:72456 IMAGE:5621698)/CN  
 E3 0 --> ALPHA 1,3-MANNOBIOSE/CN  
 E4 1 ALPHA 1,4-GALACTOSYLTRANSFERASE (HUMAN CLONE MGC:9631 IMAGE: 3913851)/CN  
 E5 1 ALPHA 1-6-GLUCOSIDASE (EC 3.2.1.70) (LACTOCOCCUS LACTIS LACT IS STRAIN IL1403 GENE DEXB)/CN  
 E6 1 ALPHA 1-ANTITRYPSIN (HUMAN)/CN  
 E7 1 ALPHA 100/CN  
 E8 1 ALPHA 1000/CN  
 E9 1 ALPHA 127/CN  
 E10 1 ALPHA 1800/CN  
 E11 1 ALPHA 1C ADRENERGIC RECEPTOR ISOFORM 2 (HUMAN CLONE P2C6)/CN  
 E12 1 ALPHA 2 ACTIN (HUMAN CLONE MGC:9221 IMAGE:3906861)/CN

=> exp alpha 1,3 mannobiose/cn

E1 1 ALPHA 1,2-MANNOSIDASE (HUMAN CLONE MGC:12553 IMAGE:3959196) / CN  
 E2 1 ALPHA 1,2-MANNOSIDASE IB (HUMAN)/CN  
 E3 0 --> ALPHA 1,3 MANNOBIOSE/CN  
 E4 2 ALPHA 1,3-FUCOSYLTRANSFERASE (HELICOBACTER PYLORI STRAIN HPA G1)/CN  
 E5 1 ALPHA 1,3-FUCOSYLTRANSFERASE FUC-T (SIMILAR TO MOUSE FUT4) ( RATTUS NORVEGICUS CLONE MGC:72456 IMAGE:5621698)/CN  
 E6 1 ALPHA 1,4-GALACTOSYLTRANSFERASE (HUMAN CLONE MGC:9631 IMAGE: 3913851)/CN  
 E7 1 ALPHA 1-6-GLUCOSIDASE (EC 3.2.1.70) (LACTOCOCCUS LACTIS LACT IS STRAIN IL1403 GENE DEXB)/CN  
 E8 1 ALPHA 1-ANTITRYPSIN (HUMAN)/CN  
 E9 1 ALPHA 100/CN  
 E10 1 ALPHA 1000/CN  
 E11 1 ALPHA 127/CN  
 E12 1 ALPHA 1800/CN

=> exp 1,3 mannobiose/cn

E1 1 1,2R,3,4S,5-PENTAAMMONIOPENTANE TETRACHLOROZINCATE TRICHLORIDE MONOHYDRATE/CN  
 E2 1 1,3 BENZENEDICARBOXYLIC ACID, POLYMER WITH 2-ETHYL-2-(HYDROXYMETHYL)-1,3-PROPANEDIOL, HEXANEDIOIC ACID, 1,6-HEXANEDIOL, 1,3-ISOBENZOFURANDIONE AND 1,1'-METHYLENEBIS(4-ISOCYANATOBENZENE), DI-ET MALONATE/CN  
 E3 0 --> 1,3 MANNOBIOSE/CN  
 E4 1 1,3 PROPANEDIOL DEHYDROGENASE (GEOBACILLUS KAUSTOPHILUS STRA

IN HTA426) /CN  
 E5 1 1,3'(2H,2'H)-SPIROBI(CYCLOPENT(B) INDOLE) /CN  
 E6 1 1,3'(2H,2'H)-SPIROBI(CYCLOPENT(B) INDOLE), 1',3,4,4'-TETRAHYDRO-1',1',3,3-TETRAMETHYL-4,4'-BIS(PHENYLMETHYL)-/CN  
 E7 1 1,3',3'-TRIMETHYL-6-NITRO-8-BROMOSPIRO(2H-1-BENZOPYRAN-2,2'-INDOLINE) /CN  
 E8 1 1,3'-(BIPYRROLIDIN)-5'-ONE, 4',4'-DIMETHYL-2'-(3-NITRO-O-TOLYL) IMINO)-1'-PHENYL-/CN  
 E9 1 1,3'-BI-1,2,4-TRIAZOLE, 3,5-DIMETHYL-/CN  
 E10 1 1,3'-BI-1,2-DICARBADODECABORANE(12) /CN  
 E11 1 1,3'-BI-1,2-DICARBADODECABORANE(12), 1'-METHYL-/CN  
 E12 1 1,3'-BI-1,2-DICARBADODECABORANE(12), 2-METHYL-/CN

=> exp alpha 1,2-mannobiose/cn  
 E1 1 ALPHA 1,2 N-ACETYLGLUCOSAMINE TRANSFERASE (NEISSERIA MENINGITidis GROUP C STRAIN FAM18 GENE RFAK) /CN  
 E2 1 ALPHA 1,2 N-ACETYLGLUCOSAMINE TRANSFERASE (SYMBIOBACTERIUM THERMOPHILUM STRAIN IAM14863) /CN  
 E3 0 --> ALPHA 1,2-MANNOBIOSE/CN  
 E4 1 ALPHA 1,2-MANNOSIDASE (HUMAN CLONE MGC:1215 IMAGE:3533651) /CN  
 E5 1 ALPHA 1,2-MANNOSIDASE (HUMAN CLONE MGC:12553 IMAGE:3959196) /CN  
 E6 1 ALPHA 1,2-MANNOSIDASE IB (HUMAN) /CN  
 E7 2 ALPHA 1,3-FUCOSYLTRANSFERASE (HELICOBACTER PYLORI STRAIN HPA G1) /CN  
 E8 1 ALPHA 1,3-FUCOSYLTRANSFERASE FUC-T (SIMILAR TO MOUSE FUT4) (RATTUS NORVEGICUS CLONE MGC:72456 IMAGE:5621698) /CN  
 E9 1 ALPHA 1,4-GALACTOSYLTRANSFERASE (HUMAN CLONE MGC:9631 IMAGE:3913851) /CN  
 E10 1 ALPHA 1-6-GLUCOSIDASE (EC 3.2.1.70) (LACTOCOCCUS LACTIS LACTIS STRAIN IL1403 GENE DEXB) /CN  
 E11 1 ALPHA 1-ANTITRYPSIN (HUMAN) /CN  
 E12 1 ALPHA 100/CN

=> exp alpha 1,6-mannobiose/cn  
 E1 1 ALPHA 1,3-FUCOSYLTRANSFERASE FUC-T (SIMILAR TO MOUSE FUT4) (RATTUS NORVEGICUS CLONE MGC:72456 IMAGE:5621698) /CN  
 E2 1 ALPHA 1,4-GALACTOSYLTRANSFERASE (HUMAN CLONE MGC:9631 IMAGE:3913851) /CN  
 E3 0 --> ALPHA 1,6-MANNOBIOSE/CN  
 E4 1 ALPHA 1-6-GLUCOSIDASE (EC 3.2.1.70) (LACTOCOCCUS LACTIS LACTIS STRAIN IL1403 GENE DEXB) /CN  
 E5 1 ALPHA 1-ANTITRYPSIN (HUMAN) /CN  
 E6 1 ALPHA 100/CN  
 E7 1 ALPHA 1000/CN  
 E8 1 ALPHA 127/CN  
 E9 1 ALPHA 1800/CN  
 E10 1 ALPHA 1C ADRENERGIC RECEPTOR ISOFORM 2 (HUMAN CLONE P2C6) /CN  
 E11 1 ALPHA 2 ACTIN (HUMAN CLONE MGC:9221 IMAGE:3906861) /CN  
 E12 1 ALPHA 2 BI-ACTIVE-ENERGIE 5/CN

=> exp mannooligosacch/cn  
 E1 1 MANNONOYL CHLORIDE, PENTAACETATE/CN  
 E2 1 MANNONOYL CHLORIDE, PENTAACETATE, L-/CN  
 E3 0 --> MANNOOLIGOSACCH/CN  
 E4 1 MANNOPENTAOSE/CN  
 E5 1 MANNOPENTAOSE SULFATE/CN  
 E6 1 MANNOPENTAOSE-DI(N-ACETYL-D-GLUCOSAMINE) /CN  
 E7 1 MANNOPEPTIMYCIN A/CN  
 E8 1 MANNOPEPTIMYCIN B/CN

E9 1 MANNOPEPTIMYCIN Δ/CN  
E10 1 MANNOPEPTIMYCIN E/CN  
E11 1 MANNOPEPTIMYCIN Γ/CN  
E12 1 MANNOPEPTIN A/CN

=> file caplus  
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FILE COVERS 1907 - 22 Jul 2008 VOL 149 ISS 4  
FILE LAST UPDATED: 21 Jul 2008 (20080721/ED)

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1 L19

1030340 THU/RL  
L20 0 L19/THU  
(L19 (L) THU/RL)

=> s 119  
L21 1 L19  
  
=> d 121 ti abs bib

L21 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2008 ACS on STN  
TI The configuration of glycosidic linkages in oligosaccharides. I.  
Application of Jackson and Hudson's oxidation method to reducing  
disaccharides  
AB Disaccharides were degraded by glycol-cleavage oxidations, on the premise  
that all disaccharides of a given class would yield the same product. The  
relative contribution of the glycosidic center to the optical activity is  
enhanced in the oxidation products, and thereby promotes a larger  
rotational difference between  $\alpha$ - and  $\beta$ - compds. than in the  
original disaccharides. All  $\alpha$ - and  $\beta$ -Me aldohexopyranosides of  
the D-series yield  $\text{OHCCH}(\text{CH}_2\text{OH})\text{OCH}(\text{OMe})\text{CHO}$  (I) by cleavage of the  
2,3,4-triol group;  $\alpha$ - and  $\beta$ -glycosides give aldehydes with  
large pos. and neg. specific rotation, resp., which differ only in  
configuration at the glycosidic center. D-Aldohexopyranose disaccharides  
having 1,6-linkages are degraded by  $\text{IO}_4^-$  (II) or  $\text{Pb}(\text{OAc})_4$  (III) to  
 $\text{OHCCH}(\text{CH}_2\text{OH})\text{OCH}(\text{CHO})\text{OCH}_2\text{CHO}$  (IV) in which the reducing end-unit has been  
converted to  $\text{HOCH}_2\text{CHO}$  and the glycosidic residue yielded a dialdehyde  
similar to I. A 1,4-hexose disaccharide such as cellobiose (V) should be  
converted by III to a structure (VI) in which the D-erythrose (VII) unit  
of the reducing end is linked at the 2-position to a dialdehyde similar to  
I, and related in configuration to I derived from  $\beta$ -D-  
aldohexopyranosides. Maltose (VIII) should yield a compound which differs  
from VI only in the configuration of the glycosidic center. All  
D-aldohexopyranose disaccharides in which the reducing end is D-glycose  
(IX) or D-mannose (X) should yield one of these two oxidation products  
depending on the configuration of the biose linkage. If D-galactose (XI)  
is the reducing end-unit, the cleavage product should be a compound similar  
to VI, in which VII is replaced by D-threose. The results obtained by  
glycol-cleavage oxidation were (disaccharide,  $[\alpha]_D$  disaccharide,  
oxidizing agent, and  $[\alpha]_D$  oxidized disaccharide given): melibiose  
(XII),  $129^\circ$ , II,  $79^\circ$ ; isomaltose (XIII),  $98^\circ$ , II,  
 $85^\circ$ ; mannobiose ( $1,6\alpha$ )  $50^\circ$ , II,  $88^\circ$ ;  
galactosido-erythritol ( $1,4\alpha$ ),  $134^\circ$ , II,  $79^\circ$ ;  
gentiobiose (XIV),  $8^\circ$ , II,  $-109^\circ$ ; mannosido-erythritol  
( $1,4\beta$ ),  $-39^\circ$ , II,  $-106^\circ$ ; VIII,  $130^\circ$ , III,  
 $24^\circ$ ; glucosido-arabinose ( $1,3\alpha$ ),  $48^\circ$ , III,  $19^\circ$ ;  
lactose (XV),  $55^\circ$ , III,  $-78^\circ$  V,  $34^\circ$ , III,  
 $-80^\circ$ ; glucosido-mannose ( $1,4\beta$ ),  $47^\circ$ , III,  $-71^\circ$ ;  
mannobiose ( $1,4\beta$ ),  $-6^\circ$ , III,  $-84^\circ$ ; glucosido-erythritol  
( $1,2\alpha$ ),  $130^\circ$  II,  $5^\circ$ ; glucosido-erythritol ( $1,2\beta$ ),  
 $-17^\circ$  II, 0; galactosido-erythritol ( $1,2\beta$ ),  $7^\circ$  II, 0;  
glucosido-fructose ( $1,5\alpha$ ),  $-8^\circ$ , II,  $36^\circ$ ; xylobiose  
( $1,4\beta$ ),  $-25^\circ$ , II,  $102^\circ$ . Disaccharides having  
1,2-linkages are over-oxidized by II and III and could not be examined by  
direct oxidation. Three such compds. were reduced to the alc., in order  
to avoid over-oxidation, and then treated with II. The products gave zero  
or very small rotations (see above). This was attributed to the formation  
of symmetrical products such as  $\text{OHCCH}[\text{OCH}(\text{CHO})\text{CH}_2\text{OH}]_2$ . It is concluded  
that the method is not applicable to 1,2- or 1,5-hexopyranose  
disaccharides. XIII and XIV were prepared from the crystalline octaacetates by  
deacetylation with  $\text{NaOMe}$ . The sirups were neutralized with  $\text{HOAc}$  (XVI),  
the  $\text{MeOH}$  evaporated, and the sirups used directly in the oxidations. In a

typical experiment, 136 mg. XII hydrate in 5 ml. H<sub>2</sub>O was mixed with 600 mg. NaIO<sub>4</sub> in 5 ml. H<sub>2</sub>O;  $\alpha$ D was almost constant for 4 hrs. at 1.26°, but dropped to 1.13° after 24 hrs. Assuming conversion of XII to 72 mg. IV, IV gave  $[\alpha]$ D 79°. Oxidations with III were carried out at 28° in a constant volume Warburg apparatus. In a typical experiment, 9 manometers were used; each flask contained 1.5 mg. VIII in 0.2 ml. 90% XVI and 25 mg. III and 10 mg. KOAc (XVII) in 1.0 ml. 90% XVI. The CO<sub>2</sub> evolved indicated the formation of 1 mole HCO<sub>2</sub>H in 5-6 hrs. The contents of all the flasks were combined, excess III destroyed by addition of 0.5 ml. (CO<sub>2</sub>H)<sub>2</sub> (XVIII) (10% in XVI), Pb(C<sub>2</sub>O<sub>4</sub>)<sub>2</sub> (XIX) filtered off and washed with XVI, and the filtrate concentrated to dryness at 40°. H<sub>2</sub>O (3 ml.) was added to the residue and the slightly turbid solution was stored for 18 hrs. at 3° to promote hydrolysis of the HCO<sub>2</sub>R groups, following which the solution was neutralized with Pb(CO<sub>3</sub>)<sub>2</sub> (XX) and filtered through Celite. The clear filtrate was used for polarimetric measurements (2-dm. tube);  $\alpha$ D 0.17° (initial) to 0.15° (24 hrs.; constant); this corresponded to  $[\alpha]$ D<sub>27</sub> 23° assuming conversion of VIII to 9.9 mg. of the  $\alpha$ -anomer of VI. Expts. with Me glycosides indicated that the loss of oxidation product using III was small. Me  $\beta$ -D-glucopyranoside gave values of  $[\alpha]$ D<sub>27</sub> -123° and -132° compared with values of -150° and -141° by oxidation with II. Larger-scale oxidations with III were carried out in glass-stoppered Erlenmeyer flasks. In a typical experiment, 51.1 mg. XV hydrate in 10 ml. 90% XVI was mixed with 750 mg. III and 300 mg. XVI in 30 ml. 90% XVI. After 3 hrs., a 2.0 ml. aliquot showed that 4.9 moles III had reacted (theory 5.0 moles). Optical rotation was determined on an aqueous solution prepared as described above;  $\alpha$ D -0.96° (initial) to -1.03° (24 hrs.; constant).  $[\alpha]$ D of VI was -76° assuming conversion of 48.6 mg. XV hydrate to 33.8 mg. VI. V, VIII, and XV hydrate were oxidized with III as described above, meso-(CHMeOH)<sub>2</sub> being used in place of XVIII, and with the omission of the use of XX. The hydrolysis rate of the formate esters of each product was measured polarimetrically. The results were (initial concentration of sugar, 1% in a 2-dm. tube at 27°) ( $\alpha$ D at 0.5, 5, 24, 48, and 72 hrs., and  $[\alpha]$ D given): VIII, 1.05°, 0.86°, 0.54°, 0.47°, 0.46°, 31°; V, 0.05°, -0.38°, -0.98°, -1.15°, -1.13°, -77°; XV, 0.10°, -0.34°, -0.86°, -1.06°, -1.06°, -73°.

AN 1957:43153 CAPLUS <>LOGINID::20080722>>

DN 51:43153

OREF 51:8016c-i,8017a-e

TI The configuration of glycosidic linkages in oligosaccharides. I. Application of Jackson and Hudson's oxidation method to reducing disaccharides

AU Charlson, A. J.; Perlin, A. S.

CS Natl. Research Council, Saskatoon, SK

SO Canadian Journal of Chemistry (1956), 34, 1804-10

CODEN: CJCHAG; ISSN: 0008-4042

DT Journal

LA Unavailable

=> s mannobiose

L22 380 MANNOBIOSE

=> file registry

COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION

FULL ESTIMATED COST

8.59 178.42

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=> s mannobiose/cn  
L23 1 MANNOBIOSE/CN

=> file caplus		
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FULL ESTIMATED COST	5.61	184.03
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FILE COVERS 1907 - 22 Jul 2008 VOL 149 ISS 4  
FILE LAST UPDATED: 21 Jul 2008 (20080721/ED)

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=> s 123/thu  
195 L23  
1030340 THU/RL  
L24 6 L23/THU  
(L23 (L) THU/RL)

=> d 124 1-6 ti abs bib

L24 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN  
TI Anti-inflammatory compositions or agents containing  $\beta$ -1,4-mannobiose  
and food or feed containing them  
AB Title compns. or agents are useful for prevention or amelioration of  
sepsis, inflammatory diseases such as rheumatoid arthritis, inflammatory  
bowel disease, etc., through suppression of IL-8 production. Thus, copra meal  
was treated with Hemicellulase GM Amano at 60° for 12 h and dried  
to water content <10%. The powder thus obtained was extracted with EtOH 3  
times, the residue was extracted with warm water at 60°, filtered, and  
the filtrate was freeze-dried to give a composition containing 21.74%  
 $\beta$ -1,4-mannobiose. The composition significantly suppressed LPS-induced  
IL-8 production by Caco-2 cells.  
AN 2008:63936 CAPLUS <<LOGINID::20080722>>  
DN 148:143661  
TI Anti-inflammatory compositions or agents containing  $\beta$ -1,4-mannobiose  
and food or feed containing them  
IN Yokomizo, Futoshi; Ibuki, Masahisa; Mine, Yoshinori; Katayama, Shigeru  
PA Fuji Oil Co., Ltd., Japan  
SO Jpn. Kokai Tokkyo Koho, 7pp.  
CODEN: JKXXAF  
DT Patent  
LA Japanese  
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
-----	----	-----	-----	-----
PI JP 2008007505	A	20080117	JP 2007-167720	20070626
PRAI US 2006-805754P	P	20060626		

L24 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN  
TI Antiallergic composition and agent, and food, beverage and animal feed  
each containing the composition or agent  
AB The invention provides an anti-allergic composition or agent comprising  
 $\beta$ -1,4-mannobiose. The composition containing  $\beta$ -1,4-mannobiose is prepared  
by degradation of mannan containing natural products derived from coconut cake,  
copra meal and palm kernel meal with mannan-degrading enzyme  
hemicellulase. The composition containing  $\beta$ -1,4-mannobiose is then dried and  
extracted with ethanol. The composition containing  $\beta$ -1,4-mannobiose has an  
inhibitory effect on mast cell degranulation. The composition containing  
 $\beta$ -1,4-mannobiose is added to foods, beverages and animal feeds.  
AN 2008:11239 CAPLUS <<LOGINID::20080722>>  
DN 148:77751  
TI Antiallergic composition and agent, and food, beverage and animal feed  
each containing the composition or agent  
IN Yokomizo, Futoshi; Ibuki, Masahisa; Mine, Yoshinori; Katayama, Shigeru  
PA Fuji Oil Company, Limited, Japan  
SO PCT Int. Appl., 18pp.  
CODEN: PIXXD2

DT Patent  
LA Japanese  
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2008001770	A1	20080103	WO 2007-JP62800	20070626
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
	RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				

PRAI US 2006-805753P P 20060626

RE.CNT 16 THERE ARE 16 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN  
TI Intestinal immunity-activating substance or agent, and food, beverage and animal feed containing the same  
AB The invention provides an intestinal immunity-activating substance or agent containing  $\beta$ -1,4-mannobiose. The intestinal immunity-activating substance containing  $\beta$ -1,4-mannobiose is prepared by degradation of mannan containing natural products derived from coconut cake, copra meal or palm kernel meal with mannan-degrading enzyme hemicellulase. The intestinal immunity-activating substance containing  $\beta$ -1,4-mannobiose is then dried and extracted with ethanol. The substance containing  $\beta$ -1,4-mannobiose enhances IgA production, has a preventive effect on diseases caused by pathogenic bacteria and viruses and also has a preventive effect on allergy. The substance containing  $\beta$ -1,4-mannobiose is added to foods, beverages and animal feeds.  
AN 2008:10173 CAPLUS <<LOGINID::20080722>>  
DN 148:77749  
TI Intestinal immunity-activating substance or agent, and food, beverage and animal feed containing the same  
IN Yokomizo, Futoshi; Ibuki, Masahisa; Mine, Yoshinori; Katayama, Shigeru  
PA Fuji Oil Company, Limited, Japan  
SO PCT Int. Appl., 17pp.  
CODEN: PIXXD2

DT Patent  
LA Japanese  
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2008001769	A1	20080103	WO 2007-JP62799	20070626
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
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BY, KG, KZ, MD, RU, TJ, TM  
PRAI US 2006-805752P P 20060626  
RE.CNT 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

- L24 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
- TI Mechanism of glycofection: enhanced nuclear import of plasmid DNA complex with disaccharide-conjugated poly(ethylenimine)s
- AB The mechanism of nuclear import of glycoconjugates consisting of disaccharide-modified polyethylenimine (PEI)/DNA polyplexes was studied using cytoplasmic microinjection. Eight reductive disaccharides (lactose, maltose, isomaltose, mannobiose, melibiose, gentiobiose, cellobiose, and laminaribiose) were conjugated with branched PEI by reductive amination. Cytoplasmic microinjection of fluorescent labeled polyplex showed that 28Lac-42Lac-PEI, and 9.3Cel-PEI showed no effect for the nuclear import of plasmid DNA. Lactose-conjugation to cationic non-viral vector increased the transfection efficiency by the enhancement to internalization of polyplex and nuclear import of gene. Nuclear import of glucopolyplex was distinct from importin- $\beta$ -dependent mechanism.
- AN 2006:887556 CAPLUS <<LOGINID::20080722>>
- DN 146:407763
- TI Mechanism of glycofection: enhanced nuclear import of plasmid DNA complex with disaccharide-conjugated poly(ethylenimine)s
- AU Nagasaki, Takeshi; Shiga, Toshiki; Jinta, Tomomi; Shinkai, Seiji
- CS Department of Bioengineering, Graduate School of Engineering, Osaka City University, Osaka, Japan
- SO PMSE Preprints (2006), 95, 667  
CODEN: PPMRA9; ISSN: 1550-6703
- PB American Chemical Society
- DT Journal; (computer optical disk)
- LA English
- L24 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
- TI Body fat-reducing compositions containing mannooligosaccharides, and foods and beverages containing them
- AB Title compns. contain mannose-based oligosaccharides with d.p. 1-10. Thus, coffee containing 1 g/100 mL mannooligosaccharide composition (prepared by high-pressure steam treatment of extraction residue of ground coffee beans) significantly reduced the size of s.c. fat area in volunteers.
- AN 2006:294389 CAPLUS <<LOGINID::20080722>>
- DN 144:330463
- TI Body fat-reducing compositions containing mannooligosaccharides, and foods and beverages containing them
- IN Asano, Ichiro; Fujii, Shigeyoshi; Muto, Katsuhito; Takao, Izumi; Ozaki, Kazuto; Nakamuro, Kenichi; Matsushima, Toshiyuki
- PA Ajinomoto General Foods, Inc., Japan
- SO Jpn. Kokai Tokyo Koho, 11 pp.  
CODEN: JKXXAF
- DT Patent
- LA Japanese
- FAN.CNT 1
- |    | PATENT NO.    | KIND | DATE     | APPLICATION NO. | DATE     |
|----|---------------|------|----------|-----------------|----------|
| PI | JP 2006083127 | A    | 20060330 | JP 2004-271412  | 20040917 |
|    | AU 2005290262 | A1   | 20060406 | AU 2005-290262  | 20050414 |
|    | CA 2580652    | A1   | 20060406 | CA 2005-2580652 | 20050414 |
|    | WO 2006036208 | A1   | 20060406 | WO 2005-US12823 | 20050414 |
- W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,  
CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,  
GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ,

LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA,  
 NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL,  
 SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA,  
 ZM, ZW  
 RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,  
 IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF,  
 CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM,  
 KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG,  
 KZ, MD, RU, TJ, TM  
 EP 1809328 A1 20070725 EP 2005-734832 20050414  
 R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,  
 IS, IT, LI, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR  
 CN 101087621 A 20071212 CN 2005-80038862 20050414  
 KR 2007083684 A 20070824 KR 2007-708570 20070416  
 IN 2007CN01566 A 20070831 IN 2007-CN1566 20070417  
 PRAI JP 2004-271412 A 20040917  
 WO 2005-US12823 W 20050414

L24 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN  
 TI Stabilized compositions of tissue-type plasminogen activator  
 AB This invention relates to a composition containing natural or modified  
 tissue-type

plasminogen activator (t-PA) and mannose derivs., for the improvement of  
 stability, solv., and half-life of t-PA. A solution was formulated containing  
 t-PA 0.5 mg/mL, NaH<sub>2</sub>PO<sub>4</sub>/Na<sub>2</sub>HPO<sub>4</sub> 0.1 M (pH 7.2), NaCl 0.8 %,  
 β-1,4-mannobiose 10 %, and Tween 80 0.01 % and freeze-dried.

AN 2001:217889 CAPLUS <>LOGINID::20080722>>  
 DN 134:242690  
 TI Stabilized compositions of tissue-type plasminogen activator  
 IN Tanaka, Junya; Yoshikawa, Genichi; Mukai, Katsuyuki  
 PA Unitika Ltd., Japan  
 SO Jpn. Kokai Tokkyo Koho, 6 pp.  
 CODEN: JKXXAF  
 DT Patent  
 LA Japanese  
 FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	JP 2001081040	A	20010327	JP 1999-256201	19990909
PRAI	JP 1999-256201		19990909		

=> file stnguide  
 COST IN U.S. DOLLARS SINCE FILE TOTAL  
 ENTRY SESSION  
 FULL ESTIMATED COST 20.06 204.09

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE	TOTAL
CA SUBSCRIBER PRICE	ENTRY	SESSION
	-4.80	-40.80

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 ENTRY SESSION

FULL ESTIMATED COST		0.12	204.21
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE	TOTAL	
	ENTRY	SESSION	
CA SUBSCRIBER PRICE	0.00	-40.80	

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FILE COVERS 1907 - 22 Jul 2008 VOL 149 ISS 4  
 FILE LAST UPDATED: 21 Jul 2008 (20080721/ED)

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This file contains CAS Registry Numbers for easy and accurate substance identification.

```
=> s methyl alpha manno?
  1060565 METHYL
  1779675 ALPHA
  58520 MANNO?
L25      64 METHYL ALPHA MANNO?
          (METHYL(W)ALPHA(W)MANNO?)
```

```
=> s nutritional or enteral or prebiotic
  66844 NUTRITIONAL
  4337 ENTERAL
  4374 PREBIOTIC
L26      74681 NUTRITIONAL OR ENTERAL OR PREBIOTIC
```

```
=> s l25 and l26
L27      0 L25 AND L26
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COST IN U.S. DOLLARS	SINCE FILE	TOTAL
	ENTRY	SESSION
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DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE	TOTAL
	ENTRY	SESSION
CA SUBSCRIBER PRICE	0.00	-40.80

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FILE CONTAINS CURRENT INFORMATION.  
LAST RELOADED: Jul 18, 2008 (20080718/UP).

=> file hcplus		
COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.06	206.96
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-40.80

FILE 'HCAPLUS' ENTERED AT 16:46:41 ON 22 JUL 2008  
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FILE COVERS 1907 - 22 Jul 2008 VOL 149 ISS 4  
FILE LAST UPDATED: 21 Jul 2008 (20080721/ED)

HCAplus now includes complete International Patent Classification (IPC) reclassification data for the second quarter of 2008.

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s gut or intestine or oral or pharmaceutical

31419 GUT		
217128 INTESTINE		
228570 ORAL		
300822 PHARMACEUTICAL		
L28 714245 GUT OR INTESTINE OR ORAL OR PHARMACEUTICAL		

=> s 125 and 128

L29 4 L25 AND L28

=> file stnguide

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	2.69	209.65
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-40.80

FILE 'STNGUIDE' ENTERED AT 16:46:43 ON 22 JUL 2008  
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FILE CONTAINS CURRENT INFORMATION.  
LAST RELOADED: Jul 18, 2008 (20080718/UP).

=> d 129 1-4 ti abs bib  
YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

L29 ANSWER 1 OF 4 HCAPLUS COPYRIGHT 2008 ACS on STN  
TI Glycoconjugate histochemistry of the digestive tract of *Triturus carnifex*  
(*Amphibia, Caudata*)  
AB In this study, the varieties of sugar residues in the gut  
glycoconjugates of *Triturus carnifex* are investigated by carbohydrate  
conventional histochem. and lectin histochem. The esophageal surface  
mucous cells contained acidic glycoconjugates, with residues of GalNAc,  
Gal  $\beta$ 1,3 GalNAc and (GlcNAc  $\beta$ 1,4)n oligomers. The gastric  
surface cells mainly produced neutral glycoproteins with residues of  
fucose, Gal  $\beta$ 1-3 GalNAc, Gal- $\alpha$ Gal, and (GlcNAc  
 $\beta$ 1,4)n oligomers in N- and O-linked glycans, as the glandular mucous  
neck cells, with residues of mannose/glucose, GalNAc, Gal  $\beta$ 1,3  
GalNAc, (GlcNAc  $\beta$ 1,4)n oligomers and fucose linked  $\alpha$ 1,6 or  
terminal  $\alpha$ 1,3 or  $\alpha$ 1,4 in O-linked glycans. The oxynticopeptic  
tubulo-vesicular system contained neutral glycoproteins with N- and  
O-linked glycans with residues of Gal- $\alpha$ Gal, Gal  $\beta$ 1-3 GalNAc and  
(GlcNAc  $\beta$ 1,4)n oligomers; Fuc linked  $\alpha$ 1,2 to Gal,  $\alpha$ 1,3 to  
GlcNAc in (poly)lactosamine chains and  $\alpha$ 1,6 to GlcNAc in N-linked  
glycans. Most of these glycoproteins probably corresponds to the  
H+K+-ATPase  $\beta$ -subunit. The intestinal goblet cells contained acidic  
glycoconjugates, with residues of GalNAc, mannose/ glucose, (GlcNAc  
 $\beta$ 1,4)n oligomers and fucose linked  $\alpha$ 1,2 to Gal in O-linked  
oligosaccharides. The different composition of the mucus in the digestive  
tracts may be correlated with its different functions. In fact the  
presence of abundant sulfation of glycoconjugates, mainly in the esophagus  
and intestine, probably confers resistance to bacterial enzymic  
degradation of the mucus barrier.

AN 2007:560386 HCAPLUS <<LOGINID::20080722>>  
DN 147:254134  
TI Glycoconjugate histochemistry of the digestive tract of *Triturus carnifex*  
(*Amphibia, Caudata*)  
AU Liquori, Giuseppa Esterina; Mastrodonato, Maria; Zizza, Sara; Ferri,  
Domenico  
CS Dipartimento di Zoologia, Laboratorio di Istologia e Anatomia comparata,  
Universita degli Studi di Bari, Bari, 70125, Italy  
SO Journal of Molecular Histology (2007), 38(3), 191-199  
CODEN: JMHOAO; ISSN: 1567-2379  
PB Springer  
DT Journal  
LA English  
RE.CNT 48 THERE ARE 48 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 2 OF 4 HCAPLUS COPYRIGHT 2008 ACS on STN  
TI Sequences of human C-type lectin DC-SIGN and DC-SIGNR, and uses thereof  
for inhibiting hepatitis C virus infection  
AB This invention provides protein sequences of human C-type lectin DC-SIGN,

DC-SIGNR, and hepatitis C virus polyprotein. This invention further provides a method of inhibiting HCV infection of a cell susceptible to HCV infection which comprises contacting the cell with an amount of a compound effective to inhibit binding of an HCV envelope glycoprotein to a DC-SIGN protein present on the surface of the cell, so as to thereby inhibit HCV infection of the cell susceptible to HCV infection. This invention provides a method of inhibiting HCV infection of a cell susceptible to HCV infection which comprises contacting the cell with an amount of a compound effective to inhibit binding of an HCV envelope glycoprotein to a DC-SIGNR protein present on the surface of the cell, so as to thereby inhibit HCV infection of the cell susceptible to HCV infection. Compds. of the present invention inhibit HCV infection of cells susceptible to HCV infection. The compds. of the present invention preferably have specificity for preventing or inhibiting infection by HCV and do not inhibit infection by other viruses, such as HIV, that may utilize DC-SIGN or DC-SIGNR for infection. Moreover the compds. of the present invention preferably do not interfere or inhibit members of the Ig superfamily, in particular, the compds. do not interfere with ICAM-2 or ICAM-3 or with ICAM-2-like, or ICAM-3-like mols.

AN 2003:5672 HCPLUS <>LOGINID::20080722>>  
 DN 138:83339  
 TI Sequences of human C-type lectin DC-SIGN and DC-SIGNR, and uses thereof for inhibiting hepatitis C virus infection  
 IN Olson, William C.; Maddon, Paul J.  
 PA Progenics Pharmaceuticals, Inc., USA  
 SO PCT Int. Appl., 166 pp.  
 CODEN: PIXXD2

DT Patent  
 LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2003000024	A2	20030103	WO 2002-US20875	20020626
	WO 2003000024	A3	20030731		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	US 20030013081	A1	20030116	US 2001-891894	20010626
	CA 2452049	A1	20030103	CA 2002-2452049	20020626
	AU 2002324461	A1	20030108	AU 2002-324461	20020626
	EP 1411980	A2	20040428	EP 2002-759107	20020626
	EP 1411980	B1	20080109		
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	JP 2005521628	T	20050721	JP 2003-506480	20020626
	AT 383170	T	20080115	AT 2002-759107	20020626
	ES 2296985	T3	20080501	ES 2002-759107	20020626
PRAI	US 2001-891894	A	20010626		
	WO 2002-US20875	W	20020626		

L29 ANSWER 3 OF 4 HCPLUS COPYRIGHT 2008 ACS on STN

TI Aromatic alpha-glycosides of mannose are powerful inhibitors of the adherence of type 1 fimbriated Escherichia coli to yeast and intestinal epithelial cells

AB Adherence of bacteria via their surface lectins to host epithelial cells is considered an important initial event in bacterial pathogenesis. Mannose-specific (type 1) fimbriae are among the most commonly found lectins in enterobacteria. The effect of aromatic  $\alpha$ -glycosides of mannose was studied on the agglutination of mannan-containing yeasts by different strains of *E. coli* and on the adherence of the bacteria to guinea pig ileal epithelial cells. In both systems, these compds. were considerably more effective inhibitors than Me  $\alpha$ -mannoside, with 4-methylumbelliferyl  $\alpha$ -mannoside and p-nitro-o-chlorophenyl  $\alpha$ -mannoside being the strongest inhibitors. Both compds. were approx. 400-times stronger inhibitors of yeast agglutination by *E. coli* O128 than was Me  $\alpha$ -mannoside and 1000- and 470-fold stronger, resp., than was Me  $\alpha$ -mannoside in inhibiting the adherence of the bacteria to ileal epithelial cells. 4-Methylumbelliferyl  $\alpha$ -mannoside was 540-1000 times more effective in inhibiting yeast agglutination by 4 addnl. strains of mannose-specific *E. coli*. It was also more efficient than Me  $\alpha$ -mannoside in removing adherent *E. coli* O128 from ileal epithelial cells. The results provide further evidence that type 1 fimbriae of *E. coli* possess a hydrophobic region next to the mannose-binding site. The results suggest that 4-methylumbelliferyl  $\alpha$ -mannoside and p-nitro-o-chlorophenyl  $\alpha$ -mannoside are good candidates for the design of therapeutic agents that may prevent adherence *in vivo* and infection by *E. coli* strains that express type 1 fimbriae.

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TI Aromatic alpha-glycosides of mannose are powerful inhibitors of the adherence of type 1 fimbriated *Escherichia coli* to yeast and intestinal epithelial cells

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L29 ANSWER 4 OF 4 HCPLUS COPYRIGHT 2008 ACS on STN

TI Transport of sugars and amino acids in the intestine: evidence for a common carrier

AB Rings of everted hamster small intestine were incubated with cycloleucine-14C (I) or L-tyrosine. D-Galactose (II) and L-arginine (III) were partially competitive inhibitors of I transport; neutral amino acids were fully competitive inhibitors. Kinetic consts. for I transport were calculated. Straight line and parabolic curves were seen with neutral amino acids and II and III, resp. I, histidine, proline, and methionine are thought to share a common binding site in the membrane. If II and III are allosteric inhibitors of the transport of neutral amino acids, probably 3 different substrate-binding sites (1 each for sugars, neutral amino acids, and basic amino acids) plus the Na<sup>+</sup>-binding site, are closely associated in the membrane, as in a mosaic. The actively transported analogs of II and III inhibited the transport of neutral amino acids. Of the basic amino acids, L-lysine was as effective as III, and L-ornithine more so. Of the sugars, II was the strongest inhibitor, but other metabolizable and nonmetabolizable actively transported sugars were inhibitory. Compds. not actively transported were inert such as methyl- $\alpha$ -glucoside, methyl- $\alpha$ -mannoside, D-fructose, L-sorbose, and 2-deoxy-D-galactose. Phlorizin was a poor inhibitor of amino acid transport, and its presence prevented the inhibitory effects of II. Nonmetabolizable sugars had qual. identical effects as II. D-Allose, with an axial --OH group as in II, was more inhibitory than glucose. For

I and tyrosine, methyl- $\alpha$ -D-mannopyranoside > D-glucose > 6-deoxy-D-glucose > 3-O-methyl-D-glucose > methyl- $\alpha$ -D-glucopyranoside > D-allose > II were inhibitors of neutral amino acid transport. II, III, glucose, and methylglucosides elicited countertransport of the neutral amino acid L-tyrosine, II > methyl- $\alpha$ -glucoside > glucose in efficiency.

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TI Transport of sugars and amino acids in the intestine: evidence for a common carrier

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